

**RECENT ADVANCES IN
ANÆSTHESIA AND ANALGESIA**

RECENT ADVANCES IN ANÆSTHESIA AND ANALGESIA

(Including Oxygen Therapy)

BY

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PREFACE TO EIGHTH EDITION

IN the first seven editions of this book it has been my aim to give a brief description of each subject followed in more detail by an account of recent advances. It has now become clear that it is no longer possible to pursue this course without producing a volume of inordinate size. In the eighth edition therefore, I have made it a working rule to consider only developments which have taken place in the past few years the reader being assumed to be familiar with earlier work. Various exceptions have had to be made to this plan as some topics are sufficiently recent to be dealt with as a whole, while others are compact enough to be treated as monographs. Examples will be found in Muscle Relaxants, Induced Hypotension and Hypothermia, Anæsthetic Explosions, the Phenothiazine Derivatives etc. Although little recent work has been done on Oxygen Therapy this chapter has been retained owing to numerous requests from readers who appear to have found it useful.

Dr J. Alfred Lee has been kind enough to share the labours of producing this volume and I am most grateful to him for his whole-hearted co-operation and wide knowledge. The disadvantages of dual authorship are considerably diminished by the fact that we found no divergence of views on any material point.

I would like to thank various individuals and firms for the loan of blocks and for permission to use illustrations from published papers and to express regret to any author whose work has inadvertently not been acknowledged.

The publishers have once again given every possible help in bringing out this edition and I am most indebted to them.

C. LANGTON HEWER

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CHAPTER I

PREMEDICATION AND SEDATION

Morphine — Pentobarbitone — Pethidine — Methylpentynol — Belladonna alkaloids — Methantheline bromide — Oxyphenonium bromide — Chlorpromazine — Hyoscyamine — Levorphan tartrate — Metapon — Heroin — Nuxetil — Nalorphine — Levallorphan tartrate — Bemegrade — Amiphenazole

ADVANCES in knowledge concerning premedication and the drugs used to achieve it have not been very numerous in recent years, and among the general sedatives it is probably correct to say that the opiates fully tested by time since they were first used as a pre-anæsthetic drug by Labbe and Guyon in 1872, are still used more widely for this purpose than any other drugs. In spite of the recent synthesis of morphine by Gates and Tscudi in 1952,¹ the organic chemist is not yet the supplier of the drug on any large scale. The great advantages of morphine in premedication are that it lessens anxiety and causes sleep. In addition it raises the threshold for pain while it alters the response to pain which is felt.² In some patients it causes vomiting but in others it never has this unpleasant effect. The emetic action is said to be due to direct stimulation of the chemoreceptor trigger zones of Borison and Wang in the medulla.³ It also exerts an anti emetic effect and sometimes prevents other emetic drugs from causing vomiting. A good review of morphine and similar drugs is that by Woolmer.⁴

The potentiation of analgesic agents. Additional evidence⁴ has been brought forward to support the theory that cholinergic or parasympathomimetic drugs potentiate the analgesic effect of morphine. A similar state of affairs is seen with the other opiates and with the synthetic analgesics pethidine levorphan and methadone⁵ so that their intensity and duration are both increased. The addition of 0.5 mg. of neostigmine to 10 mg. of papaveretum or to 2 mg. of levorphan is said to increase the analgesic effect by about 40 per cent. Similarly pyridostigmine in a dose of 1 mg. added to 2 mg. of levorphan increases the duration of pain relief of the latter drug from 6 to 12 hours.⁴⁴ The explanation of this potentiation is not yet certain. While it has been held that cholinergic drugs exert a direct analgesic effect on their own account it is probable that other mechanisms come into play also. For example parasympathomimetic drugs such as pyridostigmine and neostigmine

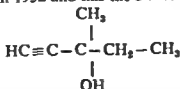
interfere with the destruction of opiates by the liver and so cause a prolongation of their action. Yet another explanation is that cholinergic substances interfere with the binding of analgesic agents to extracellular proteins and so leave larger amounts free to depress brain cells.⁴⁵ In practice the addition of pyridostigmine (1 mg) or neostigmine (0.5 mg) to the analgesic not only prolongs the action of the analgesic, but also reduces its undesirable side-effects such as intestinal stasis, bladder atony and depression of the cough reflex.

In order to overcome some of the disadvantages of morphine, especially its tendency to cause vomiting, pentobarbitone (nembutal) has been used by the intramuscular route. This drug has long enjoyed popularity when given by mouth as it causes sleep and reduces the metabolic rate quite efficiently. The veterinary solution has been used with satisfaction in human patients: it is a solution of pentobarbitone in propylene glycol, each ml containing 60 mg of drug. The usual dose is 60–90 mg given into the muscles of the buttock one hour before operation, and patients receiving this amount are usually asleep when brought to the operating theatre and remain so for three to five hours thereafter. Local irritation from the site of injection has not been reported.⁶ The ordinary intravenous solution of the sodium salt in water (7.5 per cent) can also be used for intramuscular injection.⁶ Beecher believes that the drug can act as a true analgesic.⁴¹

Pethidine has won for itself a well established place as a drug for sedative premedication. It does not cause constipation nor does it interfere with the pupillary reflexes by producing miosis. Like morphine, small doses may raise the pressure in the common bile duct and the lesser biliary ducts by inducing spasm in the sphincter of Oddi; and thus the pain resulting from biliary tract disease may be made worse. When combined with scopolamine the drugs form a very efficient injection for premedication. If popularity is to be judged by the number of synonyms, then pethidine must surely be a very widely used drug. Here are some of them: demerol, meperidine hydrochloride, isomipercaine, dolosal, dolantin, dolantal, diadone, culodat, mephudine, pantalgine, piridosal, spasmedal, adolens, antiduol, biphenal, centralgin, dispodal, dodonal, felidine, gratidin, dolvanol, lydol, operidine, precededyl, santeralgyl, sinesalgine.

Methylpentynol (Oblivon, Somnesin). This has been advertised as an agent which removes fear, apprehension and anxiety, without causing drowsiness. The dose for an adult is 250–750 mg, or 1 to 3 teaspoonfuls depending on whether capsules or elixir are preferred.

For a child between 5 and 10 years of age, 1 to 2 capsules (250-500 mg) or from 1 to 2 teaspoonfuls are advised. This new drug was first used in Britain in 1952 and has the structural formula



It is reputed to be free from undesirable side actions, is relatively non-toxic and is metabolized within two hours. It does not reduce the rate or amplitude of respiration, nor does it reduce muscle tone. Amnesia and post-operative restlessness are both absent. About fifteen minutes after taking a dose by mouth the patient becomes mildly elated and then a feeling of contentment is noticed, this lasting about one hour. It is quite suitable for ambulant patients as it does not cause disorders of behaviour, increase in reaction time, or inco-ordination. It is potentiated by barbiturates.⁷

Its chief use in premedication has up to the present been in tonsillectomy in children.⁸ In these difficult cases, the cough reflex is not abolished while recovery from anaesthesia is not prolonged. Post-operative restlessness is stated to be less troublesome than when an oral barbiturate is used. Occasionally vomiting of the drug complicates induction. For children under 8, 2 capsules, or 2 teaspoonfuls of the blue elixir are recommended; for those over 8, 3 capsules, or 3 teaspoonfuls of elixir. Scopolamine has been added to the oral solution to avoid the needle prick.⁴⁸

In general therapeutics methylpentynol has been used as a hypnotic where insomnia is due to anxiety or nervous tension,⁹ before a dreaded visit to the dentist,¹⁰ before minor painful procedures such as catheterization, certain radiological examinations, minor operations, etc.¹¹ Some success has followed the use of the drug to relieve anxiety and fear during labour, the mechanism of which does not appear to be harmfully altered. Foetal respiratory function likewise is not depressed. The social uses of methylpentynol include the relief of anxiety in stammering, in travel sickness and before public appearances, interviews or oral examinations (e.g. the F.F.A.R.C.S. or the D.A.). Toxic effects have been reported.⁴⁹

The belladonna alkaloids remain unchallenged as drugs for inhibiting the secretion of saliva and of mucus from the upper respiratory tract. Like its allies the antihistamines, atropine causes slight drowsiness. Accommodation is not altered by a dose of

gr $\frac{1}{100}$ (0.6 mg) The undoubted fact that atropine helps to prevent laryngospasm and bronchospasm during anaesthesia with thiopentone and cyclopropane is difficult to explain, as the skeletal muscles in the larynx are not theoretically influenced by the drug. Impulses from the aortic and carotid pressor receptors are blocked by atropine and so is the intestinal contraction associated with sympathetic block in extradural and subarachnoid analgesia. It is doubtful if atropine lessens the incidence of cardiac arrhythmias during induction of anaesthesia.¹² These were formerly thought to be mediated through the vagus, but the amount of atropine used clinically is insufficient to block the peripheral action of the vagus on the heart while the present view is that the nerve impulses causing arrhythmias during anaesthesia are mediated at any rate on the efferent side, by the sympathetic.¹³

Because atropine was not always satisfactory as a drying agent—perhaps because of inadequate dosage Burstein¹⁴ advocated the use of methantheline bromide (banthine) for this purpose the dosage suggested being 1 mg for each year of age. In his hands this proved to be a safe and efficient inhibitor of secretions. This opinion was supported by Alver and Wood,¹⁵ using slightly smaller doses, and moreover they showed it reduced the incidence of vomiting. On the other hand Ruben¹⁶ showed that the beneficial drying effect had to be paid for with an increase in the pulse rate making the agent undesirable for routine use. Oxyphenonium bromide (antrenyl) is a synthetic benzylic ester with an anti cholinergic effect on organs supplied by the parasympathetic nervous system, similar to atropine. It has been used with success in the United States to inhibit salivation before anaesthesia and recently other workers¹⁷ have shown experimentally and clinically that it is a useful substitute for atropine, having a slightly longer duration of effect and being a little more potent than the same dose of atropine. It provides a relatively safe means of obtaining optimum anti cholinergic effects which last for several hours. It is without subjective side effects, but is not active as a drying agent when given by mouth.

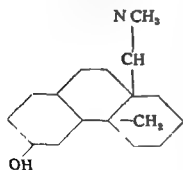
In a laboratory controlled investigation, the salivary depressant activity of atropine was compared with that of an equal dose (gr $\frac{1}{100}$) of hyoscyamine (bellafoline) and the latter was found to be more efficient but tachycardia and other side effects were considered to be undesirable.¹⁸ In thyrotoxic patients it has been shown that tachycardia is rather less when scopolamine is used than it is after atropine.¹⁹ The biphasic effect of atropine on the heart described in classical pharmacology is probably seen only when the

drug is given subcutaneously. When administered by the intravenous route there is evidence¹⁷ that the slight tachycardia due to block of the vagal effects on the S-A node is not preceded by a bradycardia from stimulation of the vagal nuclei in the medulla. If this is so then atropine and neostigmine can safely be injected from the same syringe when it is desired to reverse the effects of a non depolarizing relaxant.

Chlorpromazine (largactil, thorazine) has been used for sedative premedication as for most other purposes in medicine. Boulton¹⁸ in a controlled clinical trial gave the drug by mouth before and after operation to patients undergoing thoracic operations. A 25 mg tablet was given the night before, on the morning of operation, after operation and thrice daily the next day. It was found to allay anxiety and as a probable consequence, vomiting was less frequent than in the control series. It was suggested that in abdominal cases the post operative doses might be given per rectum in the form of a 100-mg suppository, a dose recently increased to 300 mg. With pethidine 100 mg, 50 mg of chlorpromazine injected intramuscularly 1 hour before operation has given good results with extradural block¹⁹ but it may cause rather severe hypotension.

Sedatives

Levorphan tartrate (laevo-methorphan, levorphanol, l-dromoran) is laevo 3 hydroxy N methyl morphinan and was synthesized by



Schneider and Grassner in 1949. The laevo isomer is twice as active as the racemic drug (dromoran). (The dextro-isomer, dextro-methorphan (romilar), is used to quell coughing.) It is effectively absorbed when given by mouth, and the ordinary dose is 1.5 to 2 mg. The duration of action is 8-10 hours. Its chief use is to relieve pain, and in severe cases 4 mg may be required. Although it does

not depress the cough reflex it may cause intestinal spasm and so is not the ideal drug for the relief of pain due to colic. As a pre-anæsthetic drug it lacks two essential attributes, it does not allay anxiety nor does it promote sleep¹⁸. On the other hand it causes less hangover than does morphine when used as a post-operative sedative. It is available in 2 mg tablets and 2 mg ampoules.

Metapon is a synthetic derivative of morphine, it is methyl dihydromorphinone. The drug is supplied in 3 mg. capsules for oral administration and two such capsules are said to be equal in analgesic potency to morphine gr $\frac{1}{4}$ (16 mg). It is superior to morphine in that it causes no nausea, no vomiting, and very little respiratory depression, on the other hand it is a poor hypnotic. Its chief use is in the relief of severe chronic pain but it is a powerful drug of addiction and has no place in premedication.

It is proposed that diacetyl morphine (heroin) should no longer be manufactured or sold in Britain. This decision arrived at by the Ministry of Health after consultation with certain officials of the medical profession was made at the request of the government of the United States, as heroin addiction is a considerable social evil in that country and it was thought that its suppression here would cut down supplies elsewhere. A vocal section of medical opinion in Britain is strongly opposed to the disappearance of this useful drug, and in anaesthesia it eases post-operative pain without causing as much nausea as morphine and is most useful as a supplement to nitrous oxide and oxygen and thiopentone. It quietsens the upper respiratory tract reflexes without causing very marked respiratory depression.*

Nisentil (alphaprodine). This drug, a congener of pethidine is di alpha 1, 3-dimethyl-4-phenyl-4 propionoxypiperidine. It has been used intravenously to supplement thiopentone nitrous oxide oxygen anaesthesia in place of pethidine than which it is said to act rather more rapidly and to have a shorter duration of effect. With it an increased controllability of the depth of anaesthesia without undue depression of respiration can be achieved provided the dose is kept small.²⁰ In obstetrics in doses of 40 mg. subcutaneously, it has proved to be a suitable agent for use during labour because of the rapid onset of its action, the lack of maternal and foetal side-effects and the short duration of the sedation produced. This last attribute makes it a useful agent in rapid labours.²¹ Its use in premedication²² about 40 mg. intramuscularly and in post-operative sedation²³ has been favourably reported on. The recommended dose is 20-30 mg. by vein and 40-60 mg. by mouth or intramuscularly.

Nisentil given intravenously raises the pressure of the cerebro-spinal fluid and pethidine has a similar effect. If levallorphan is given at the same time as the nisentil, this does not occur. After pethidine however nalorphine has a much better action than has

* Sanction to manufacture this drug has recently been extended for a further year.

levallorphan and should be given in the ratio of 1 to 20. This may be specially important in neurological surgery where it might well be desirable to prevent this rise in cerebrospinal fluid pressure without at the same time interfering with the other actions of the pethidine.⁴⁷

Nalorphine. This drug is N allyl normorphine and is also known as nalline or lethidrone. It differs from morphine in having an allyl (C_3H_5) instead of a methyl (CH_3) radical attached to the nitrogen atom. While the antagonistic effect on the respiratory depression due to morphine was described as long ago as 1915 by Pohl,²⁴ it was not until 1944 that interest in the drug was revived by Hart and McCawley.²⁵ It was used in anaesthesia in 1952 by Eckenhoff and his colleagues.²⁶ Nalorphine reverses the respiratory depressant effect of morphine and its congeners such as heroin, codein, dilaudid and metapon, and also that of pethidine, dromoran and methadone.

Narcotized and non narcotized patients react differently to the drug. When given to unpremedicated subjects it has a similar effect though slightly less marked to morphine and causes drowsiness, hallucinations and some depression of breathing. It is a poor analgesic and does not cause the spastic immobility of the bowel seen after morphine. Administered to a narcotized patient nalorphine abolishes analgesia, euphoria, respiratory depression, anti diuresis,⁴⁸ miosis and hyperglycaemia due to morphine. The lessening of narcosis is neither very marked nor sustained. On the alimentary canal it abolishes the state of spasticity and the absence of peristalsis caused by morphine. It restores reflexes and raises lowered blood pressure. The EEG pattern is altered from that of deep sleep to that of the waking state. The duration of its effects is shorter than that of morphine. It is not absorbed from the alimentary canal and is administered by either intravenous or intramuscular injection. As side-effects, nausea, dysphoria, and sweating are prominent, so that it is totally unsuitable for use in ambulatory patients. Its clinical effects come on within two to three minutes of the intravenous injection of 3-5 mg and may disappear within ten minutes. A close watch must be kept on the blood pressure which tends to rise, and only if there is great respiratory depression should repeated doses be given.²⁷ If respiratory depression is not a marked feature of the patient's clinical state, then the injection of nalorphine may increase sedation and decrease ventilation.

The dosage of the drug is critical, if insufficient is given the opiate or depressant is not displaced from the cell receptors whereas

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The dosage of the drug is critical, if insufficient is given the opiate or depressant is not displaced from the cell receptors whereas

if too much enters the circulation although the opiate is displaced sufficient nalorphine remains to produce its own narcotic effects. A dose of 4-5 mg is said to antagonize 50 mg of pethidine or 5 mg of morphine, so that the normal amount suitable for an average adult is 10 mg and that for a neonate, 0.2 to 0.5 mg.

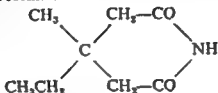
Bodman states²⁷ that nalorphine can be useful in the treatment of poisoning by opiates in anaesthesia, in general medical cases, in chronic emphysematous patients and those with right heart failure. It may be helpful if used to reverse the sedative effect of morphine given to undiagnosed acute abdominal emergencies, and it may find a place in the diagnosis of addiction to morphine, pethidine etc. When it is given to an addict severe withdrawal symptoms are produced. The patient sweats, his pupils dilate, he breathes rapidly and becomes agitated. This is the morphine abstinence syndrome in an acute form and it comes on half to three-quarters of an hour after the injection. In midwifery nalorphine has had some triumphs. It has been given to rather heavily morphinized patients just before delivery with a view to reversing the depressant effects on the foetal respiratory function. The drug can also be given in 0.2 mg doses—cautiously repeated if necessary—into the umbilical vein of babies with asphyxia neonatorum²⁸ which is perhaps better than giving it to the mother. It has been successfully used to relieve spasm of the sphincter of Oddi induced by morphine or pethidine²⁹.

Levallorphan tartrate (lorfan) is the N allyl derivative of laevo-methorphan (l-dromoran) to which it bears the same relationship as does nalorphine to morphine. It, too, antagonizes the depressant effects of morphine, dromoran, pethidine, methadone etc., but not their analgesic effects, and so has been used to reverse the hypopnoea due to these agents supplementing nitrous oxide oxygen anaesthesia, without interfering with their analgesic action. It has also been used in combination with narcotics for pre-operative medication for the relief of post-operative pain and in obstetrics. Suggested dosage ratios are pethidine:levallorphan 80:1, morphine or nisential:levallorphan 50:1, levorphan:levallorphan 10:1. It potentiates the depressant properties of pentobarbitone. Neither nalorphine nor levallorphan should be used to counteract the respiratory or general depression which may be seen after overdose of barbiturates, cyclopropane or the volatile anaesthetics.

Two new drugs have recently been introduced for the treatment of barbiturate poisoning, morphine poisoning and the reversal of barbiturate anaesthesia. These are bemegride (megimide, N P 13) and amiphenazole (daptazole). Amiphenazole was introduced by

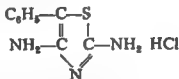
Davies and his colleagues in 1949³⁰ and was investigated as a morphine antagonist by Shaw and Bentley³¹ The two drugs were used together in the treatment of coma following poisonous doses of the barbiturates by Shulman in 1955,³² while the use of bemegride alone to reverse barbiturate anaesthesia was reported by Harris in 1955³³

Bemegride is β ethyl β methyl glutarimide, a crystalline compound with the structural formula



It is sparingly soluble in water

Amiphenazole is 2,4-diamino-5-phenylthiazole hydrochloride with the structural formula



It is too insoluble in water but such solutions do not last long. The crystalline powder is quite stable.

Bemegride reduces by about 50 per cent the sleeping time of rats following dosage with a barbiturate and is effective when given intravenously, intramuscularly or by mouth. It stimulates respiration depressed by barbiturates, restores the electroencephalogram to normal in subjects under barbiturate narcosis and has a large safety margin. Gross overdosage will cause convulsions.³⁴

The pharmacology of amiphenazole has been studied by Shaw and Shulman³⁵ and it has been shown that dogs made comatose by morphine are awake within two minutes of the intravenous injection of a therapeutic dose of the drug. Respiratory stimulation precedes arousal. It is active when given either into a vein or into a muscle. Animals made unconscious with barbiturates do not respond to the drug when used alone but it is specifically effective in relieving the respiratory depression caused by large doses of opiates, pethidine and methadone. It appears to stimulate the depth of respiration rather than the rate and has little effect on the analgesic effects of these agents unless given in large amounts. Such large amounts may raise the blood pressure.

Shaw and Shulman³⁵ are of the opinion that morphine should be used more freely than is now the case, its depressant effects being

prevented by amiphenazole. Patients with severe pain should be well and properly relieved by amounts of morphine in the order of 1 or 2 gr 6 or 8 hourly in $\frac{1}{4}$ gr increments until the desired effect is obtained, each $\frac{1}{4}$ gr dose being accompanied by 12.5 mg of amiphenazole intramuscularly. If the respiratory rate becomes too slow i.e. less than 6 or 8 per minute, then 10 mg of the drug can be given at 10-minute intervals up to a total of 50 mg.

In the treatment of barbiturate intoxication amiphenazole and bemegride are used together in the manner described by Shulman, Shaw and their associates³⁶. The drugs are given into the tubing of a dextrose drip, the bemegride as a 0.5 per cent solution the amiphenazole as a 1.5 per cent solution two separate syringes being employed. One ml of amiphenazole is followed by 10 ml of bemegride and these injections are repeated at 5 minute intervals until the upper respiratory tract reflexes and the muscle tone show signs of returning. Increasing activity of the plantar withdrawal reflex is said to be a good indication of response of the patient to the treatment. In severe cases as much as 200 ml of bemegride (1 gm) and 20 ml of amiphenazole (300 mg) may be required over a period of 2 hours.

Following anaesthesia with an intravenous barbiturate a rapid return to consciousness has followed the intravenous injection of 50 mg of bemegride as described by Harris³⁷. A number of patients so treated fall asleep again but can be roused by strong stimuli. Side effects are rare and so far no contra-indications have been noted.

The drugs are supplied as follows. Megimide in 100 ml ampoules of 0.5 per cent solution for the treatment of barbiturate poisoning and in 10-ml ampoules of the same strength for the rapid reversal of barbiturate anaesthesia. Daptazole comes in 300 mg ampoules of dry powder which should be dissolved in 20 ml of saline for the treatment of barbiturate intoxication. For administration with morphine 15 mg of dry powder in an ampoule is to be dissolved in 1.5 ml of saline. These solutions must be thrown out after 12 hours as they are relatively unstable.

References

1. GATES N. and TSCUDI R. 1952 *J of Amer Chem Soc*
2. HARDY J. H., WOLF H. G., and GOODELL, H. 1940 *J clin Invest* 19 649
3. BORISON H. L. and WANG S. L. 1953 *Pharmacol Revue* 5 193
4. WOOLMER R., 1955 *Brit J Anzsth* 27 267 (Jan)
5. JARVIS, J. R., 1953 *Ohio St med J.*, 49 308
6. BEUTNER, H. K., 1951 *Anesthesiology* 12 863
7. TROTTER, P., 1954 *Lancet* 2 1302.

- 8 RENDALL, CHRISTINE M 1954 *Brit med J* March 13 641
- 9 DAVIS N 1954 *Brit med J* March 20 701
- 10 SIMMONS, H J 1954 *Brit Dent J* July 6 21
- 11 BOAG A G *Brit med J* 1954 Feb 13 393
- 12 BOURNE, G., 1954 *Lancet* Sept 11 522
- 13 GOODMAN L S and GILMAN A 1955, *The Pharmacological Basis of Therapeutics* 2nd ed 42 Macmillan
- 14 BURSTEIN C L 1953 *Anesthesiology* 14 567 (Nov)
- 15 RUBEN H 1955 *Anesthesiology* 16 653 (July)
- 16 SARNOFF, E J COPE O 1954 *Anesthesiology* 15 484
- 17 HUNTER, A R 1953 *Brit med J* 1, 640 (March 21)
- 18 BOULTON F H 1955 *Anaesthesia* 10 233 (July)
- 19 SLOVKA M H *et al* 1951 *J Pharmacol* 101 33
- 20 SILER E S, FOLDES, F F *et al* 1954 *Brit J Anaesth* 26 405 (Nov)
- 21 ENICH J E (Jr) 1955 *Amer J Obstet Gynec* 69 124 (Jan)
- 22 BELINKOFF S 1955 *Anaesth & Analges* 34 116 (March-April)
- 23 BAGIRACH E H GODHOLM A N and BETCHER A M 1955, *Surgery* 37 44 (March)
- 24 POHL, J 1915 *Z exper Path Ther* 18 370
- 25 HART E R and McCRAWLEY E L 1944 *J Pharmacol* 82 339
- 26 ECKENHOFF, J E, HOFFMANN G L, and DRIFFS R D 1952 *Anesthesiology* 13 242.
- 27 BODMAN R I 1953 *Proc R Soc Med* 46 933
- 28 ECKENHOFF J E. *et al* 1953 *Amer J Obstet Gynec* 65 1269
- 29 SCHAPIRO H and BEAL, J M 1953 *Surgery* 34 870
- 30 DAVIES W, MACLAREN, J A and WILKINSON L R 1949 *Med J Aust* 2 869
- 31 SHAW F H and BENTLEY G 1952, *Nature* 169 712
32. SHULMAN A *et al* 1955 *Brit med J* 1 1238
- 33 HARRIS T A B 1955 *Lancet* 1 181
- 34 SHAW F H *et al* 1954 *Nature* 174 403
- 35 SHAW F H and SHULMAN A 1955 *Brit med J* 1 1367
- 36 SHULMAN A, SHAW F H, CASS N M and WYFFE H M, 1955, *Brit med J* 1 1238
- 37 MUSHIN W W and ADAMS A S 1955 *Brit J Anaesth* 27 519 (Nov)
- 38 ALVER E C and WOOD J M V 1956 *Anesthesiology* 17 73 (Jan)
- 39 HAYERS L 1956 *Brit J Anaesth* 28 83 (Feb)
- 40 GALLOON S 1956 *Brit J Anaesth* 28 113 (March)
- 41 COHEN E M and BEECHER H K 1951 *J Amer med Ass* 147 1664
- 42 SLAUGHTER D and CROSS E G 1938 *J Pharmacol* 63 34
- 43 SLAUGHTER D 1950 *Anaesth & Analges* 29 34 (Jan-Feb)
- 44 OEHLANDT G 1955 *Med Klinik* 50 2202
- 45 KNOLL, J *et al* 1953 *Acta physiol hung* 4 131
- 46 PRESCOTT F 1956 *Canad Anesth Soc J* 3 39 (Jan)
- 47 SWERDLOW M 1956 *Anaesthesia* 11 149 (April)
- 48 GUSTERSON F R 1956 *Proc World Congress Anesthesiologists* (1955) 43 Burgess Minneapolis
- 49 MARLEY E and CHAMBERS J S W 1956 *Brit med J* 2 1467 (Dec 22)

CHAPTER 2

INHALATION ANÆSTHETICS

Diethyl Ether — Fluorinated Derivatives — Methyl N Propyl Ether—Nitrous Oxide—Ethylene—Cyclobutane—Cyclopropane—Chloroform—Trichlorethylene—Xenon—Ethyl Vinyl Ether

Diethyl Ether

WHILE ether is probably one of the most widely used anæsthetic agents in the world and is certainly one of the safest, there is not at present much original work being undertaken in connection with it. This is perhaps not unexpected when it is remembered that the literature covering it stretches back for well over one hundred years.

J D Laycock,⁴⁹ while paying tribute to the usefulness of the signposts along the path to anæsthesia erected by Arthur Guedel in 1937 emphasizes that with modern agents and special muscle relaxants these stages and planes are no longer as helpful as they once were. He further points out that according to W B Primrose intercostal paralysis occurring in Guedel's third and fourth planes is anatomically non-existent.⁵⁰

To overcome these difficulties in teaching anæsthesia Laycock has suggested a different system. Stage 1 is consciousness with disorientation and analgesia. Stage 2 is unconsciousness with reflex activity. Stage 3 unconsciousness with reflex depression and Stage 4 respiratory paralysis.

Guedel's classification has also been altered by Harris.⁵¹ In this classification Stage 1 is associated with depression of the higher centres and corresponds to Guedel's classifications Stages 1, 2 and 3, Plane 1. Stage 2 is the depression of sensory co-ordination and corresponds to Guedel's classification Stage 3 Plane 2. Stage 3 is the stage of depression of areas of motor co-ordination and corresponds to Guedel's Stage 3 Planes 3 and 4. Stage 4 is the depression of the medulla and corresponds to Guedel's classification Stage 4. It consists of failure of the respiratory centre, vasomotor and cardiac centres.*

J F Artusio¹ has sought to separate the first stage of ether narcosis into three planes and in the deepest of these he claims to produce a true state of analgesia but one in which cerebration is excellent and the patient is comfortable and co-operative while

THE STANDARD SEQUENCE OF THE DEPRESSION OF THE BRAIN BY BLOOD BORNE ANAESTHETICS*

STAGES OF ANAESTHESIA

13

HARRIS'S CLASSIFICATION		GUEDEL'S CLASSIFICATION	
Depression of the higher centres	Amnesia	Stage 1	Analgesia
	Co-operative stupor	Stage 2	Delirium
	Non-co operative stupor	Plane 1	Sleep
	Anaesthetic sleep	Plane 2	Sensory loss
Depression of areas of sensory co-ordination	Loss of the ability to react to external stimulus	Stage 3 (Surgical anaesthesia)	Loss of muscle tone
Depression of areas of motor co-ordination	Loss of muscle tone in { Small muscle groups Large muscle groups Intercostal muscles	Plane 3	Intercostal paralysis
Depression of the vital medullary centres	Failure of the respiratory centre	Plane 4	Medullary paralysis
	Failure of the vasomotor centre		
	Failure of the cardiac centre		

* *The Mode of Action of Anaesthetics* 1951 by T A B Harris (E & S Livingstone Ltd Edinburgh)

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no great abdominal relaxation, have a special application to obstetric cases as a low blood ether level in the fetus can be obtained

Ether vapour managed as a true gas has been very favourably reported on.¹¹ A technique is described in which activated charcoal is employed to produce the vapour, the use of which enables the anaesthetist to have a fine control over the partial pressure of the vapour delivered to the patient. Reflexes from the upper respiratory tract are said to be less troublesome than when ether is given by the orthodox techniques. A similar technique was of course described by members of the Oxford school in 1941 using the Oxford Vaporizer Number 2

Fluorinated Derivatives

In an effort to remove the danger of fire and explosion from the use of ether, experiments have been conducted with trifluoroethyl vinyl ether.¹² This substance which is non flammable, gives promise of future use and is said to equal in potency and have a similar anaesthetic effect to ether. It causes no anatomical or physiological changes in the liver and is without serious action on the heart. Free fluorine is not released from the compound in the body

Later work with this agent places the lower limit of flammability at 4 per cent in air or oxygen diethyl ether being flammable at a concentration of about 2 per cent.¹⁷ In dogs the drug appears to have a protective effect on the myocardium when adrenaline is administered. On the upper respiratory tract it is not as likely to cause coughing and bucking as is ether. A striking effect is its action on the blood pressure which can be significantly lowered even at light planes of anaesthesia. In the United States trifluoroethyl vinyl ether is known as fluoromar.^{18, 19}

Yet another fluorinated volatile agent has been used successfully to provide anaesthesia in humans and this is 2 bromo 2 chloro 1 1 1 trifluoroethane (CF_3CHClBr). It is known as halothane (fluothane) and is a colourless liquid with a sp g of 1.86 and a boiling point of 50.2°C . It is not decomposed by warm alkalis such as soda lime, but can be disintegrated by strong light. It is four times as potent as diethyl ether and can be used alone for the induction of anaesthesia in 2 to 4 per cent concentration and for its maintenance in about 1 per cent concentration. As the result of a careful clinical trial the following results have emerged.²⁰ It appears to be a powerful depressant of sympathetic activity with the power to abolish the shock syndrome. Stimulation of the

surgical procedures are taking place. He suggests that this is a good method of pain relief during operations for the correction of congenital cardiac malformations.

It is well known that ether anæsthesia causes hyperglycæmia and that this is probably due to impulses which arise in the midbrain and descend along the splanchnic nerves to the suprarenal glands causing the liberation of adrenaline. This hormone then liberates glycogen from the liver into the blood stream. Certain adrenergic blocking agents, such as certain alkaloids of ergot, have been shown to inhibit the hyperglycæmia² when given in non toxic doses. On the other hand, priscoline and dibenamine in reasonable doses did not show this effect.³ Ether hyperglycæmia is partially inhibited if anæsthesia is induced with thiopentone.³

The renal blood flow is considerably reduced during ether and cyclopropane anæsthesia but a return to normal takes place soon after the termination of the anæsthesia.⁴ This is neurogenic in origin and is abolished by renal denervation.⁵ Ether anæsthesia has no effect on the extensibility or elastic recoil of muscle fibres although it exercises a depressant effect on the central nervous system, on peripheral nerve conduction and on the myoneural junction.⁶

The respiratory stimulating effects of ether are often forgotten in these days when respiratory depression caused by commonly used agents can so readily be counteracted by intermittent positive pressure. In practice, however, this depression is too often allowed to persist for a long period. Even in Stage 3, Plane IV of ether anæsthesia alveolar ventilation is stimulated and causes a mild respiratory alkalosis.⁷ It has been shown that certain ill and sensitive patients can be carried to a plane of anæsthesia with a blood ether concentration which would not be sufficient to produce a corresponding level of anæsthesia in a fit and normal individual.⁸ It appears that disulfuram (antabuse) does not potentiate the central nervous system depressant effects of ether and chloroform, although it does potentiate the action of barbiturates in animals.⁹

Investigations have been made on the concentration of ether in the blood of intubated and of non intubated patients and it has been shown¹⁰ that when carbon dioxide is used as a respiratory stimulant to facilitate rapid intubation deep ether anæsthesia is not required for oral intubation. With a cuffed endotracheal tube in place anæsthesia can be maintained with a lower blood ether concentration than if the patient is not intubated assuming in both cases that the anæsthesia is kept as light as possible but sufficient to avoid vomiting, straining and coughing. These findings made on patients requiring

opinion in question. Severe bone marrow aplasia leading to grave and fatal neutropenia after prolonged anaesthesia with nitrous oxide during the treatment of tetanus, would seem to be a new and unexpected danger. So far three deaths have been reported, from Denmark and from Australia. Ordinary medical nitrous oxide was used and it is of course possible that an impurity hitherto unsuspected was the cause of the toxic effects when the gas was inhaled for periods up to four or five days or longer. The bone marrow changes are said to disappear when inhalation of the gas is discontinued⁶¹ so it is unlikely that one of the other drugs used concurrently such as d tubocurarine or a sedative was to blame. At the time of writing therefore, a warning must be given that nitrous oxide should not be used for long periods in the treatment of tetanus, bulbar poliomyelitis etc. The treatment of fainting in the dental chair by lowering the patient's head has been stressed by Bourne⁶².

When nitrous oxide is given with oxygen, unsupplemented with other drugs, mild hypoxia is an ever-present danger. It can be diagnosed by the oxygen depression test¹⁵. If a few breaths of pure oxygen are given to a patient who is suffering from oxygen lack a sudden and of course temporary fall in systolic blood pressure occurs. This is said to be more reliable than the oxygen apnoea test which is carried out in a similar way and was described by McKesson many years ago.

An innovation in technique for the administration of nitrous oxide in the dental chair which has been given the name of amnalgnesia, has recently been described^{62, 63}. The method is noteworthy because it allows 15 per cent or 20 per cent of oxygen to be used for the duration of the procedure with the exception of a short period at the beginning of induction. Amnalgnesia has been described⁶⁴ as a plane of surgical anaesthesia between Guedel's stages 1 and 2 wherein operations may be performed without pain or the memory of it. It thus comes between analgesia and delirium. When used for children, anaesthesia is induced with pure gas⁶⁵ but after five or six breaths 15 per cent oxygen is added. In an average case forty breaths of this mixture are required to get the patient into the amnalgnesic state. The right time to allow the dental surgeon to begin his extractions is when the breathing is free and regular when the eyelids are relaxed and the eyeballs have lost their expression and are either moving from side to side or else are fixed in a squint. At this point the patient is free from cyanosis and there is neither stertor nor jactitation. The surgeon need not be hurried as the stage of amnalgnesia can be maintained for long periods. Slight

secretion of the salivary, mucous and bronchial glands is not seen while nausea and vomiting are rare. Cardiac arrhythmias of the vagal type are frequent in unatropinized but abolished in atropinized patients. Cardiovascular collapse shown by bradycardia and hypotension tends to follow the combined use of d tubocurarine and controlled respiration in patients who are under the influence of the drug whereas the substitution of suxamethonium for d tubocurarine did not have this untoward effect. Post operative diuresis can occur which might be harmful to patients in a state of electrolyte imbalance. In 500 cases no operative deaths were seen. In anæsthetic concentrations in oxygen and air, its vapour is said to be non flammable. Further information about these fluorine-containing hydrocarbon volatile anæsthetics will be awaited with interest by all anæsthetists who work with surgeons addicted to the use of diathermy.

Methyl-N Propyl Ether

This agent does not seem to have gained greatly in popularity during recent years although it has its select band of enthusiastic adherents. Post operative analgesia which may last for 9 to 12 hours has been reported¹³ while hypotension is likely to be produced if during the induction period the vapour strength is too rapidly increased. An increase once deeper anæsthesia has been established does not have this effect.

Nitrous Oxide

This useful gas is more firmly established in modern anæsthetic practice to day than ever before. When given so that the patient receives at least 20 per cent of oxygen it becomes a most useful and safe agent for producing analgesia. In the absence of hypoxia its effects are almost universally regarded as harmless and when combined with a muscle relaxant and with thiopentone and pethidine, it is an excellent and very widely used gas. On the other hand when it is used as the sole anæsthetic agent in robust patients its weak properties demand that it should be combined with less than 20 per cent of oxygen in order to secure control of the patient. In these circumstances the associated hypoxia may on occasion cause serious complications and irreversible neurological changes¹⁴. What cannot be cured had better be prevented.

If there is one opinion on which all anæsthetists will agree it is that nitrous oxide when given with sufficient oxygen is completely free from toxic symptoms. Recent reports^{14, 15} however call this

opinion in question. Severe bone marrow aplasia leading to grave and fatal neutropenia after prolonged anaesthesia with nitrous oxide during the treatment of tetanus, would seem to be a new and unexpected danger. So far three deaths have been reported, from Denmark and from Australia. Ordinary medical nitrous oxide was used and it is of course possible that an impurity hitherto unsuspected was the cause of the toxic effects when the gas is inhaled for periods up to four or five days or longer. The bone marrow changes are said to disappear when inhalation of the gas is discontinued⁶¹ so it is unlikely that one of the other drugs used concurrently such as d tubocurarine or a sedative was to blame. At the time of writing therefore a warning must be given that nitrous oxide should not be used for long periods in the treatment of tetanus, bulbar poliomyelitis etc. The treatment of fainting in the dental chair by lowering the patient's head has been stressed by Bourne.⁶²

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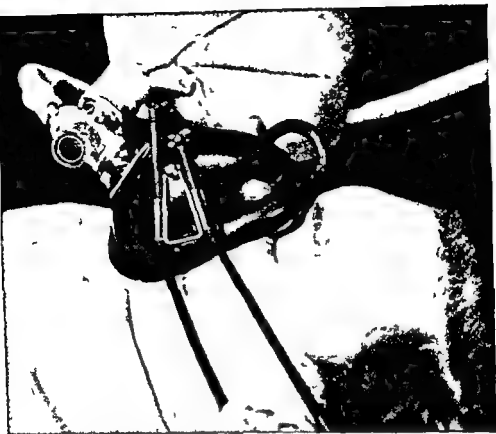


FIG 1 Method of Reducing Deadspace (the endotracheal tube is connected directly to gas inlet) (Brennan, H J *Anæsthesia*)

Changes in the blood gases associated with various methods of induction of anæsthesia preparatory to endotracheal intubation have been studied by Lachman and his colleagues⁴³ They found that the greatest alteration in the blood levels of oxygen and carbon dioxide occurs during a slow induction with ether or cyclopropane to a plane allowing an easy intubation The changes are less when a rapid induction is carried out using an intravenous barbiturate and a relaxant These blood gas changes can however be reduced to a minimum if before the barbiturate and relaxant are given the patient is asked to hyperventilate with pure oxygen for a short period

An interesting phenomenon which has been called diffusion hypoxia has been described⁴ It is well known that some patients become cyanotic during the time immediately following anæsthesia with gas and oxygen despite good ventilation During this time the oxygen saturation of the blood may fall to 90 per cent, while the

movement of the patient as he is stimulated may be seen, but is of no consequence so long as the expression does not return to his eyes. In adults, rather more oxygen can be allowed. Premedication is unnecessary and recovery is uneventful. As always in the dental chair, attention must constantly be given to the maintenance of a free airway with avoidance of air leaks and mouth breathing.

At the present time the passage of anæsthetic and analgesic drugs across the placental barrier is receiving widespread study. Nitrous oxide, as would be expected, passes over very readily into the foetal bloodstream where it is found in approximately 58 per cent of the concentration existing in maternal blood.¹⁸

Nitrous oxide is such a weak anæsthetic that its tension in the lungs must be as high as possible, compatible with adequate oxygenation. This condition is never obtained when the ordinary semi open continuous flow machines are used. Ruben¹⁷ reports his good results using nitrous oxide and adequate oxygen, and curare without any supplementary volatile or intravenous analgesics or hypnotics. Great care is taken to see that the highest possible partial pressure of nitrous oxide is presented to the lungs together with a 20 per cent moiety of oxygen. Dead space is kept to a minimum by the use of an endotracheal tube, leakage is avoided and rebreathing as far as possible eliminated by the use of a non return valve close to the patient. Intermittent positive pressure respiration by increasing the ventilation and the partial pressure of gas in the lungs aids the technique. If adequate doses of a muscle relaxant are now given a very satisfactory anæsthetic sequence results, with the patient able to walk back to the ward very soon after the conclusion of the operation. A somewhat similar method was described by Brennan¹⁶ who advocated induction of anæsthesia with a smallish dose of thiopentone combined with a muscle relaxant. If it is desired to pass an endotracheal tube this is connected to a special combined bag and expiratory valve mount which projects through the facepiece. The fitting allows a leak proof joint to be made at the face with the mask so avoiding the use of a cuffed tube or of a pharyngeal pack. In addition, it prevents inflation of the stomach. With the tube in position the patient is mildly hyperventilated for about 10 minutes before the skin incision is made, and anæsthesia is continued using 2 litres of oxygen and 6 of nitrous oxide each minute in the same way. In the majority of cases the patient does not react to the incision nor does he react again during the operation. Occasionally a small dose of pethidine is required to maintain tranquillity but this happens rarely.

dental analgesia, but there is no doubt that the patient tends to hang round the surgery, feeling out of sorts, for a longer time than when a well given gas oxygen anæsthetic has been used. It is seen at its best in nitrous oxide resistant patients, while its administration demands much less skill than does a smooth gas oxygen anæsthesia. In major surgery the use of equal volumes of cyclopropane and oxygen for induction of anæsthesia is a most useful method of ensuring rapidity. In the handicapped patient little if any hypotension is caused as may be the case with a rapid induction by an intravenous barbiturate. If now, after twenty or thirty breaths of the mixture, a suitable injection of suxamethonium is given, endotracheal intubation with a cuffed tube can be very rapidly carried out and regurgitation of undigested gastric contents rendered very unlikely. A slight head up tilt is an additional help in these cases.

H. R. Griffith, one of the pioneers of the use of cyclopropane, has written of his experiences during the administration of 20,000 cases.²¹ He regards the gas as potent, non irritating, comparatively non toxic and rapidly eliminated, allowing for the use of plenty of oxygen. He looks on it as a suitable agent for use in poor risk cases, and emphasizes the following points: (1) Respect its potency but do not be afraid of it. (2) Never give cyclopropane unless an endotracheal tube and laryngoscope are available because of the possibility of respiratory obstruction due to laryngeal spasm. In such cases, the injection of suxamethonium will both aid intubation and may by releasing the spasm, remove the need for it. (3) Leave the endotracheal tube in place after operation until purposeful movements return, because of the possibility of extubation spasm and the hypoxia it may cause. (4) Keep the depth of anæsthesia as level and smooth as possible. (5) Ensure proper ventilation. This last is of paramount importance. Assisted breathing which usually results in apnoea when cyclopropane is being given and becomes controlled breathing, must be used in the great majority of administrations, so depressing is the gas to the respiratory centre. (6) Worry about tachycardia.⁸ Griffith warns that the sudden rise in heart rate to 120 per minute or above may be a danger sign pointing to the onset of ventricular fibrillation and calls for a reduction in the tension of gas being offered to the patient. He regards arrhythmia as relatively unimportant, an opinion shared by most administrators of experience. (7) Use such other agents with the gas that prudence and convenience suggest, such as a small dose of an intravenous barbiturate for induction and a muscle relaxant for ensuring a loose abdomen. (8) Be intelligently explosion conscious, especially as

nitrous oxide is being rapidly eliminated from the lungs. This gas may form 10 to 12 per cent of the total expired volume and the outward diffusion of nitrous oxide lowers the partial pressure of the oxygen in the alveoli. The hypoxia so produced may cause cardiac arrest in old patients or those with cardiac or pulmonary disease. An oxygen-enriched atmosphere should thus be given in the 10 to 15 minutes after the cessation of a long nitrous oxide oxygen anæsthetic. The accident of diffusion hypoxia is all the more likely to occur if in addition there is laryngeal spasm, hypoventilation or mild respiratory obstruction.

Ethylene

This gas has never been much used in Britain although it was introduced as an anæsthetic in man more than three decades ago⁶³. Its present use is greatest in the southern and mid western regions of the United States but even in these areas it does not appear to be gaining in popularity.

Cyclopropane

In the ten year period following 1935, cyclopropane gained considerably in popularity at the expense of ether, but during the past decade it in turn has given place to the intravenous barbiturate, gas oxygen, relaxant, pethidine sequence partly due to the increased use of diathermy by surgeons. It has always had its supporters however, and recently a slight upward trend in its use has been in evidence. Bourne⁶⁴ finds the gas very practical in the out patient department for such operations as dental extractions, and has designed an ingenious piece of apparatus for this purpose which is portable, neat and inexpensive⁶⁵. He uses an ordinary facepiece gives a dozen or so breaths of a mixture of the gas with oxygen from a 6-litre bag and should it be necessary, continues the administration from a nasal inhaler using at least 20 per cent oxygen with nitrous oxide. Vomiting is rather frequent after the use of the gas but can be lessened if the patient sucks a tablet of hyosine half an hour beforehand. Originally a mixture of equal parts of cyclopropane and oxygen was used but it was pointed out that this might explode if a spark was produced from the friction of the dental forceps on the enamel of a tooth. To overcome this danger the pure oxygen was later replaced by a mixture of equal volumes of oxygen and nitrogen²⁰ metered through an oxygen rotameter the three gases used in this way become non flammable. When this technique is employed the dangers of hypoxia are removed from

results following the combination of these two agents in 45 patients, many being poor surgical risks on whom according to the author of the report, operation would have been impossible without the benefits of the vasopressor. In this series no dangerous signs or symptoms developed. The association of these two drugs should however be regarded with some suspicion, and the substitution for noradrenaline of phenylephrine might be a more logical procedure. Whatever may be the case with noradrenaline, there is no doubt that adrenaline will very readily induce undesirable cardiac effects in subjects under cyclopropane anaesthesia.

The electro-cardiographic effects of the injection of 0.5 to 2 mg of neostigmine and 0.8 mg of atropine sulphate on patients under cyclopropane anaesthesia have been studied²⁸. Classical cholinergic reactions were produced such as sinus bradycardia, wandering pacemaker, auriculo-ventricular block and sinus arrest, and they were potentiated by the gas. There was no evidence that the atropine potentiated the cholinergic effects of neostigmine by central stimulation; instead it caused an opposite adrenergic effect with cardiac irritability, multifocal ventricular extrasystoles, etc. This adrenergic activity could, during cyclopropane anaesthesia, lead to ventricular fibrillation and together with cardiac inhibition is a possible cause of death in patients who are subjected to neostigmine during cyclopropane anaesthesia.

The electroencephalographic pattern of patients under cyclopropane anaesthesia has been investigated⁹ and six levels of anaesthesia have been recognized. These bear a direct relationship to increasing blood cyclopropane concentrations. It has always been thought that the effect of cyclopropane on the liver is more benign than that of ether, but recent investigations³⁰ would tend to show that the difference between their effects is not significant. One of the disadvantages attending the use of the gas is the tendency it has to cause bronchospasm in asthmatics and in some apparently normal patients. If care is taken to introduce the gas very gradually, this complication may be avoided. In fact cyclopropane has been recommended³¹ as an efficient and safe mode of treatment for status asthmaticus. A slow prolonged induction carries the patient down to the third stage of anaesthesia where he remains for 15 to 30 minutes. The treatment may have to be repeated. Intramuscular injection of 5 ml of paraldehyde to which a little hyaluronidase may with advantage be added, has been used successfully to quieten the patient who develops delirium during emergence from cyclopropane anaesthesia. Pitocin, because it is not completely free from

regards static electricity Griffith regards the dangerous area round a cyclopropane administration as never more than a few inches away from the patient's face the mask or the machine (9) Pay most careful attention to the patient at all times. If this excellent body of advice is followed, cyclopropane deserves perhaps to be used more in the future than it has been in the recent past. This opinion is reinforced by the report of another large series, this time of 40 000 administrations². For the treatment of the various types of cardiac arrhythmia arising during the administration, desaturation is advised while heart disease itself is not regarded as a contra indication to the use of the gas which maintains its high reputation despite the disadvantages associated with its use such as its flammability and its tendency to cause hypotension in the immediate post operative period, and occasional increased capillary oozing during operation.

Does the agent in fact cause more bleeding in the wound than other comparable agents? Opinions have differed on this point for twenty years. McLoughlin²³ has shown that in Stage 3, Planes 1 and 2 of cyclopropane anæsthesia, bleeding is directly proportional to depth while in all planes oozing is reduced if a proper interchange of gases prevents hypercapnia. His suggestion for minimizing bleeding with the gas is to avoid the lightness of Plane 1 and to use assisted or controlled breathing in every case. The increase in cardiac output, in comparison with that caused by ether, together with the passively dilated vascular field, may be a causal factor. On the other hand the work of Price and his colleagues²⁴ shows that during cyclopropane anæsthesia there is an increase in both the central venous and the arterial blood pressure which is made greater by respiratory acidosis and by hypoxia. These findings suggest that the gas may act by reducing the competence of the heart, while simultaneously augmenting the return of blood from the periphery.

Post-anæsthetic hypotension following the use of cyclopropane—and other agents too—seems to bear a definite relationship to hypercapnia, as suggested some years ago by Dripps²⁵. Fresh work gives further evidence²⁶ suggesting again that in most cases the hypotension is due to the change from hypercapnia to hypocapnia at the end of the anæsthetic the carbon dioxide excess being due to underventilation the result of the depressant effect of the gas on the respiratory centre. Here is additional support for the view that really efficient ventilation is obligatory during anæsthesia.

Is it wise to give a continuous noradrenaline drip to a patient receiving cyclopropane? A recently published paper²⁷ describes the

in the use of the drug, on the other hand they do not agree with the marked fear with which at present the drug is regarded. Before its use they suggest that respiratory sedatives such as opiates and barbiturates should be avoided. They confirmed that it has a direct depressant effect on the myocardium and a reflex effect on cardiac rhythm, the former being the more common. In addition it can markedly depress vasomotor tone, although a progressive fall in blood pressure with the passage of time was not seen. A sudden increase in concentration of the inhaled vapour, as following a deep breath, was seen to cause myocardial depression.

In a final summary of the conclusions reached, Waters emphasizes the need for apparatus capable of changing the concentration of the vapour by 0.1 per cent as the drug is extremely potent. There is always need for additional oxygen, while induction should be slow and gradual. Very light planes of anaesthesia are often capable of giving good operating conditions. Inhalation of a high concentration of vapour suddenly may act on the myocardium, causing a precipitous hypotension perhaps accompanied by cardiac arrest. This may occur during induction, as the vapour is unirritating. A second and more common type of depression is due to vasomotor paralysis, but with an exact and constant control of the concentration of vapour inhaled, a normal blood pressure can be maintained for long periods. Should hypotension be necessary to aid the surgeon a controlled increase in vapour strength can safely be used to procure this effect. Relative overdosage is an ever present risk, a risk made worse by the lack of irritating properties of the drug. Waters concludes with a warning that administration of chloroform to be safe demands the greatest possible attention on the part of the anaesthetist who must be physically and mentally fit and alert, must have a finger constantly on the patient's pulse, must be able to vary the vapour strength at will and have a fine control over it, must give oxygen and finally, must be competent to ventilate the patient with oxygen artificially to reduce the blood vapour tension when necessary. It must be pointed out that in their series the Wisconsin workers noted 4 cases of cardiac arrest, 1 required cardiac massage and the remaining 3 responded to removal of the vapour and oxygen inflation of the lungs.

Trichlorethylene

As the years go by, this drug becomes more and more firmly established as a safe non irritating non flammable, volatile agent for the production of analgesia and the lighter planes of anaesthesia.

pitressin the pressor principle, should not be given to a patient under cyclopropane anæsthesia on account of the possibility of causing an attack of coronary arterial spasm

Cyclobutane

Experiments with this gas, the next higher homologue of cyclopropane, have been carried out by Vandam and Dripps³ Induction with it often proved to be stormy and recovery from anæsthesia prolonged, while the incidence of cardiac arrhythmia was high From the results of this work it seems doubtful if the drug holds much promise as a clinical anæsthetic agent

Chloroform

The shady reputation of this famous old drug continues in spite of a notable effort on the part of a team of workers from the State of Wisconsin General Hospital at Madison, headed by Ralph Waters²³ These investigators set about to explore the uses and misuses the advantages and the dangers of chloroform as if it was a new drug introduced for the first time From serial observations of the icteric index, cephalin cholesterol flocculation test, bromsulphalein retention test, thymol turbidity reaction and prothrombin time made before and after operation in a series of 65 patients who were anæsthetized with chloroform and a similar control group who received some other anæsthetic agent the following results emerged Slight hepatic dysfunction was seen in 52 per cent of the patients who received chloroform and in 44 per cent of the control group The abnormalities were mild and disappeared within ten days Clinically 1 of the chloroform group and 3 of the controls became slightly jaundiced No delayed chloroform poisoning was seen in either series This serious lesion however is related to the nutrition of the patient who receives the anæsthetic and in contrast to the Wisconsin workers, Lunt reports three such cases²⁴

Kidney function was tested in a series of 40 patients who received chloroform and 40 patients given other anæsthetics No significant differences in post operative renal function as tested by urine analysis non protein nitrogen estimations urea clearance tests and phenol sulphophthalein excretion were demonstrated between the two groups Blood sugar and alkali reserve determinations likewise showed no notable differences when one group was compared with another

The effects of chloroform on the heart and cardiovascular system were most carefully studied and as a result of these investigations the Madison workers certainly do not advocate a widespread revival

spontaneous cardiac irregularity in dogs under the influence of nitrous oxide oxygen and trichlorethylene, in the absence of overdosage with the last drug. Cases of sudden cardiac arrest are however occasionally reported in patients anaesthetized with trichlorethylene,⁴⁷ and a very interesting example of such a catastrophe has been described⁴⁸ interesting because the young patient, a United States marine was inhaling the vapour from a Cyprane inhaler set at figure 7 using the technique of self administration when he collapsed because of ventricular fibrillation and required 45 minutes of cardiac massage before a normal heart beat was re established. He made a partial recovery. Nine cases of primary cardiac failure, possibly due to trichlorethylene are mentioned in the report of 1000 deaths associated with anaesthesia recently brought to the attention of the Association of Anaesthetists.⁴¹ Such incidents are a warning that full resuscitative measures should always be at hand when even a so-called minor anaesthetic is given.

Tachypnoea occurring during trichlorethylene anaesthesia has been the subject of an investigation by Dundee.⁴⁹ In a series of careful clinical tests he found that the symptom occurs in about one third of all patients who receive the drug and is more common at the extremes of age. The larger the dose of an intravenous barbiturate used for induction of anaesthesia the less the likelihood of the patient developing tachypnoea. But the opposite view to this has also been advanced.⁴⁶ Rapid breathing during trichlorethylene anaesthesia should always be regarded as being due to a relative overdose in the particular patient. Intravenous injection of 20 mg doses of pethidine will usually retard the rate of breathing if reduction of the vapour tension offered to the patient is not practicable. If allowed to persist tachypnoea reduces the effective tidal volume and causes a reduction in the blood oxygen level.

Self administered inhalation of trichlorethylene for a period of 10-15 minutes has been reported to give relief from pain lasting several hours in patients suffering from advanced malignant disease.⁵

Xenon

The first report on this rare gas used as an anaesthetic agent was contributed by Cullen and Gross in 1951.⁴¹ A later communication⁴ finds its potency to be at least equal to that of ethylene, while it causes no respiratory depression slight bradycardia and no cardiac arrhythmia. Biochemical disturbances were minimal in the small series of cases described. In another investigation dealing with the

In the United States, where in its early days it was looked on with some suspicion as a kind of different coloured chloroform, it is gaining rapidly in popularity.

The drug shows few signs of toxicity either acute or chronic, and Nowill³⁵ exposed a batch of different animals to its vapour in a strength between 0.05 and 0.1 vol per cent for periods averaging 18 hours daily for 3 months. This prolonged exposure caused no observable functional or anatomical abnormality. Additional evidence of the benign nature of trichlorethylene is provided by the investigations of C. C. Richards and L. Bachman,⁴⁵ who show that 3 hours of anæsthesia with trichlorethylene administered to 5 dogs under controlled conditions of oxygenation and nutrition, and given to maintain a level of anæsthesia comparable to surgical anæsthesia, had no effect upon liver function as measured by the bromsulphalein retention test on the first, third, fifth and fourteenth post operative days. The same test under similar conditions using chloroform had an adverse effect on liver formation. It has been known for some time⁴⁷ that a portion of the trichlorethylene inhaled is converted to trichloroacetic acid and is excreted in the urine. This may be harmful if it is found in the urine in amounts greater than 7.5 mg per 100 ml. High urinary volumes lead to more speedy elimination of trichloroacetic acid and so does a high metabolic rate. On the other hand, infections and obesity sometimes delay elimination which may be a causative factor in aggravating post operative electrolyte imbalance.⁴⁸

The analysis of small concentrations of trichlorethylene vapour by interferometry has been described.³⁶ With a Zeiss interferometer, concentration of vapour as low as 0.5 per cent can be measured with great accuracy, using the refractive index of the gas mixture for the tests. The concentration of several vaporizers and inhalers has been calibrated by this method. For dental work a concentration of 0.05 to 0.1 per cent is adequate for most cases combined with nitrous oxide and oxygen.⁴⁸

Trichlorethylene addiction has been reported.³⁷ It may lead to psychosis. More work has been done on adrenaline induced cardiac irregularities in the dog during anæsthesia with trichlorethylene, cyclopropane, chloroform and ethyl chloride.³⁸ It was found that trichlorethylene sensitized the dog's heart to adrenaline as much as, or more, than did cyclopropane. Chloroform caused less sensitization to adrenaline than did either cyclopropane or trichlorethylene. Ethyl chloride had a similar effect to cyclopropane and trichlorethylene. This work again brings to light the infrequency of

muscular relaxation is required but may prove useful to induce anaesthesia rapidly and to maintain it in a light plane. There is said to be a wide margin of safety between surgical anaesthesia and respiratory arrest. Recovery is rapid and sequelae are few. It would appear that its use should be restricted to the production of light anaesthesia,⁴⁴ in which case it enters the field at present occupied by trichlorethylene, vinylene, neothyl, etc.

References

- 1 ARTUSIO J F 1954 *J Pharmacol* 111 343 (July)
- 2 WATTS D T 1954 *Curr Res Anaesth* 33 343 (Sept)
- 3 BASS W P *et al* 1953 *Anesthesiology* 14 19 (Jan)
- 4 DE WARDENER H E 1955 *Anaesthesia* 10 18 (Jan)
- 5 MILES H E *et al* 1952, *J Physiol* 118 140 (Sept 26)
- 6 GRAHAM J D P *et al* 1952 *Brit J Anaesth* 24 168 (July)
- 7 GABBARD J G *et al* 1952 *Ann Surg* 136 680 (Oct)
- 8 HILL, F W *et al* 1952 *Anaesthesia* 7 243 (Oct)
- 9 GRUBER C M *et al* 1954 *Arch Int Pharmacology* 97 79 (Feb)
- 10 MACKENZIE A PASK, E A and ROBSON J G 1954 *Brit J Anaesth* 26 111 (March)
- 11 BRANCH D R 1953 *Curr Res Anaesth* 32 217 (July)
- 12 KRANTZ J C *et al* 1953 *J Pharmacol* 108 488 (Aug)
- 13 BARNETT M S 1954 *Anaesthesia* 9 153 (July)
- 14 COURVILLE, C B 1955 *Anesth & Analges* 34 61 (March-April)
- 15 HARRIS J E. 1954 *Anesth & Analges* 33 398 (Nov-Dec)
- 16 COHEN E N *et al* 1953 *Surg Gynec & Obstet* 97 456 (Oct)
- 17 RUBEN H 1953 *Brit J Anaesth* 25 227 (July)
- 18 BRENNAN H J 1952 *Anaesthesia* 7 27 (Jan)
- 19 BOURNE J G 1952 *Lancet* 2 705 (Oct 11)
- 20 BOURNE J G and MORTON H J V 1955 *Lancet* 1 20
- 21 GRIFFITH H R. 1953 *Anesth & Analges* 32 23 (Jan-Feb)
- 22 VOLPITTO P P 1954 *J med Ass Georgia* 43 201 (March)
- 23 McLOUGHLIN G 1954 *Brit J Anaesth* 26 84 (March)
- 24 PRICE H L *et al* 1953 *Anesthesiology* 14 1 (Jan)
- 25 DRIPPS R D 1947 *Anesthesiology* 8 15 (Jan)
- 26 BUCKLEY J J *et al* 1953 *Anesthesiology* 14 226 (May)
- 27 SURKS S N *et al* 1954 *Surgery* 35 104 (Jan)
- 28 JACOBSON E *et al* 1954 *Anesthesiology* 15 407 (July)
- 29 POSSATI S *et al* 1953 *Anesth & Analges* 32 130 (March-April)
- 30 FRENCH A B *et al* 1952 *Ann Surg* 135 145 (Feb)
- 31 BENTOLILA L 1951 *Ann Allergy* 9 519 (July-Aug)
- 32 VANDAM L and DRIPPS R W 1955 *Anesthesiology* 16 48 (Jan)
- 33 WATERS R H ed 1951 *Chloroform—A Study after One Hundred Years*
Univ of Wisconsin Press
- 34 LUNT R L 1953 *Brit med J* 1 489 (Feb 28)
- 35 NOWILL, W K. *et al* 1954 *Anesthesiology* 15 462 (Sept)
- 36 HALL, K. D 1953 *Anesthesiology* 14 38 (Jan)
- 37 O'CONNOR W A 1954 *Brit med J* 2 451
- 38 MORRIS L E *et al* 1953 *Anesthesiology* 14 153 (March)
- 39 BERNSTINE M L 1954 *Arch Surg Chicago* 68 262 (Feb)
- 40 DUNDER J W 1953 *Brit J Anaesth* 25 3 (Jan)
- 41 CULLEN S C and GROSS E D 1951 *Fed Proc* 10 290
- 42 FINK H R 1955 *Anesthesiology* 16 511 (July)
- 43 LACHMAN R J *et al* 1955 *Anesthesiology* 16 29 (Jan)

influence of xenon oxygen and nitrous oxide-oxygen mixtures on *in vitro* oxidation of guinea pig brain tissue, it was found that, using 80:20 mixtures oxygen uptake was suppressed but no significant difference was noticed between the xenon oxygen and the nitrous oxide-oxygen mixtures. When this non potent gas is inhaled at an elevated pressure it can cause profound anæsthesia which is not accompanied by hypoxia provided that the partial pressure of oxygen in the respired atmosphere is 200 mm. of mercury or above. Under these conditions the quantity of xenon in the arterial blood is directly proportional to the partial pressure of the gas in the respired atmosphere. Appreciable electroencephalographic activity persists during deep anæsthesia with xenon.⁴⁴ In a series of experiments on rabbits it was found⁴⁵ that the corneal reflex is abolished when over 80 per cent of xenon is inhaled while at 70 per cent there is no response to stimulation of a leg muscle. It is of interest that this gas which is inert chemically can produce a significant pharmacological effect. Xenon-oxygen mixtures are of course non flammable.

Ethyl Vinyl Ether (EVE)

This volatile liquid anæsthetic is chemically intermediate between divinyl and diethyl ether and has the formula $C_2H_5-O-C_2H_3$. Its use was first suggested by Leake and Chen in 1930⁵⁴ and later Krantz⁵⁵ experimented with it on animals. The drug which is known commercially as vinamar is a clear nearly colourless liquid stabilized with phenyl alpha naphthylamine. It has a pungent odour which some people find unpleasant, a vapour density of approximately 2.5 and it boils at a temperature of 35.8° C. Commercially it is prepared by the action of acetylene on ethyl alcohol under pressure in the presence of potassium ethylate as a catalyst. It is decomposed by sunlight and its vapour is flammable in air and oxygen.

Ethyl vinyl and divinyl ether vapour should not be allowed to come into contact with large surfaces of skin prepared with iodine, as acetaldehyde and iodoacetaldehyde the latter a powerful lachrymator may be set free.⁵⁷

As an anæsthetic it behaves more like divinyl than diethyl ether. It has been responsible for short convulsive episodes readily controlled by oxygen inflation when given to infants on an open mask under which carbon dioxide has been allowed to accumulate. Its toxic effects on the liver are not pronounced while its actions on the cardiovascular system in light planes of anæsthesia are benign.⁵⁶ It is not the drug to use on its own in robust adults in whom profound

CHAPTER 3

APPARATUS FOR INHALATION ANÆSTHESIA

Apparatus for General Anæsthesia—Trilene Vaporizers—Dental Apparatus—Cyclopropane Apparatus—Machines for Artificial Respiration—Respiratory Valves—The Semi-closed Circuit—Elimination of Carbon Dioxide from Anæsthetic Circuits—Apparatus for Infants and Children—Gas Analysis—Antisepsis of Apparatus—Electronic Aids

IN this very technically-minded age new apparatus for making inhalation anæsthesia more efficient, if not more simple, is constantly making its appearance. The anæsthetist loves gadgets and many are the ingenious novelties which have appeared in the journals devoted to the specialty in recent years. In such a book as this it has been quite impossible to describe them all, but a representative selection has been made which will it is hoped either whet the appetite or cause further frustration according to the temperament of the reader.

There is no doubt that in the recent past many anæsthetists have tolerated far too high a tension of carbon dioxide in their anæsthetic circuits and it is gratifying that considerable research has been devoted to the rectification of this error. In recent years nearly all

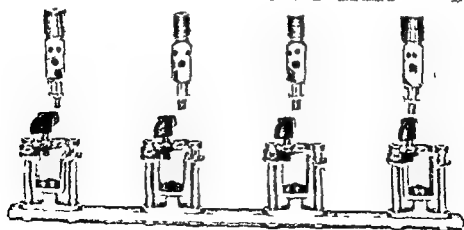


FIG 2 New non interchangeable valve system for medical gas administration (British Standard 1319) (*Anæsthesia*)

- 44 PITTINGER, C II *et al.*, 1953 *Anesthesiology* 16 551 (July)
- 45 RICHARDS C C and BACHMAN L 1955 *Curr Res Anæsth* 34 307
(Sept-Oct)
- 46 GILCHRIST, E and GOLDSMITH M W 1956 *Anæsthesia* 11 28 (Jan)
- 47 FRANT R. and WESTENDORF J 1950 *Arch Indust Hyg* 1 308
- 48 BOSTON F K 1956 *Anæsthesia* 11 37 (Jan)
- 49 LAYCOCK J D 1953 *Anæsthesia* 8 15 (Jan)
- 50 PRIMROSE W B 1952 *Brit J Anæsth* 24 3
- 51 HARRIS T A II 1951 *The Mode of Action of Anæsthetic Drugs* 217
Edinburgh E & S Livingstone
- 52 DILLON J II 1956 *Anesthesiology* 17, 208 (Jan)
- 53 LUCHHARDT A B and CARTER J B 1923 *J Amer med Ass* 80 765
- 54 LEAKE C D and CHEN M Y 1930 *Proc Soc exp Biol N Y* 28 151
- 55 KRANTZ J C *et al* 1947 *J Pharmacol* 109 88
- 56 DORNETTE W H L and ORTH O S 1955 *Anæsth & Analges* 34 26
(Jan-Feb)
- 57 WHITCHER C *et al* 1956 *Anesthesiology* 17 503 (May)
- 58 SADOVE M S *et al* 1956 *Anesthesiology* 17 591 (July)
- 59 GAINZA E *et al* 1956 *Brit J Anæsth* 28 411 (Sept)
- 60 JOHNSTONE MICHAEL 1956 *Brit J Anæsth* 28 392 (Sept)
- 61 EDWARDS G MORTON H J V PASK E A WYLIE W D 1956
Anæsthesia 11 194 (July)
- 62 KLOCK J H 1955 *Anæsth & Analges* 34 379
- 63 TOM A 1956 *Brit med J* 1 1085 (May 12)
- 64 LASSEN H C A *et al* 1956 *Lancet* 1 527 (April 28)
- 65 WILSON P MARTIN F I R LAST P M 1956 *Lancet* 2 448 (Sept 1)
- 66 BOURNE J G personal communication (in the press)
- 67 NORRIS W and STUART P 1957 *Brit med J* 1 860 (April 13)
- 68 BOURNE J G 1957 *Lancet* 2 499 (Sept 14)

absorption can be selected by the rotation of a knob. The absorption canister is made of glass and so coloured soda lime can be used. A spare canister with cover is available to facilitate change to fresh soda lime when necessary. The ether vaporizer has been re designed and will ensure a gradual and steady increase in the concentration of ether vapour. The reservoir bag has been placed just above the soda lime canister and the trichlorethylene vaporizer has a safety interlock coupled to the absorber, which prevents contamination of soda lime with trichlorethylene vapour. The draw over technique can be used if the reservoir bag control is turned to air. The

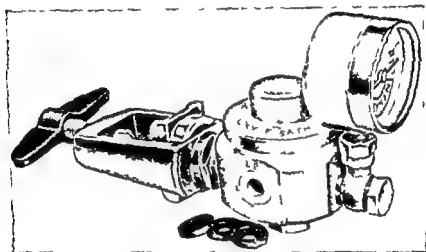


FIG 4 Reducing valve (Type A S) (Airmed Ltd)

resistance to breathing is 4 mm of water with normal minute volumes. The firm of Airmed Ltd, who make the new Marrett Head have also introduced a new regulator for medical gases, known as the type A S. This small regulator is made of light metal and no rubber or perishable material is used in its construction. It reduces the pressure of gases to 25 lb per square inch.

During the war the original Oxford Vaporizer served a most useful purpose both at home and abroad. This has now been followed by an improved model known as the EMO Inhaler for ether anaesthesia⁴⁷. It was developed at the Nuffield Department of Anaesthesia in the University of Oxford, and has replaced the older Oxford Vaporizer. It has been designed to deliver any desired concentration of anaesthetic vapour irrespective of changes in the temperature of the liquid anaesthetic, throughout the range likely to be encountered in clinical practice. No hot water is required for

anæsthetic apparatus in use in Britain has been made safer from the risk of explosion by the incorporation in them of anti static rubber, while in the near future, incorrect coupling of cylinders of anæsthetic gases will become physically impossible when the proposed alterations in anæsthetic machines are carried out⁴⁸ See Fig 2

The modern treatment of bulbar poliomyelitis and of severe tetanus by tracheostomy and intermittent positive pressure respiration has had its influence on the design of anæsthetic apparatus, especially in connection with automatic devices for the maintenance of artificial respiration. The approval, under certain conditions by the Central Midwives Board of the self administration of trichlor ethylene has also led to the introduction of several pieces of apparatus suitable for this purpose

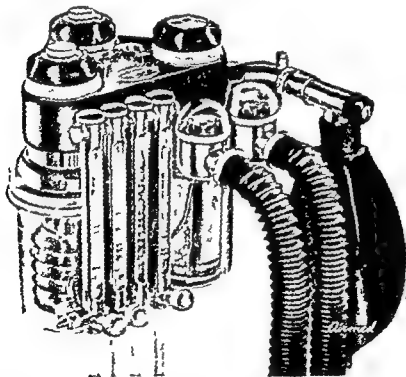


FIG 3 Marrett anæsthetic head TFC model

The new Marrett Anæsthetic Head TFC Model to and fro and circle absorber is an improvement on the old model. This is claimed to possess all the advantages of its predecessor with the following improvements: Circle or to-and-fro carbon dioxide

extent of normal respiration. Rebreathing is prevented by unidirectional valves. The Radcliffe Respiration Pump can be used with it if mechanical inflation is required (see page 48).

The Department of Anaesthesia at Cardiff has devised the Cardiff Inflating Valve which can be adapted for use with the Oxford Inflating Bellows.¹ Before the bellows can be connected to an endotracheal tube some interposed device is required which will ensure that the patient's lungs will empty quickly to the outer air at the end of each inflation. When the bellows are compressed the expiratory outlet is blocked and the patient's lungs are inflated easily. As soon as the end of the inspiratory cycle is reached the pressure within the chest, helped by a light spring in the valve, results in uncovering of the expiratory outlet so that the patient's lungs can empty easily to the outer air. Artificial respiration can thus be carried out with air. It is obtainable from the Pentland Instrument Company, George Street, Oxford.

To facilitate assisted breathing P. M. Mehta⁴⁵ has modified the Langton Hewer face mask for goitre operations by machining out a disc of metal in the top of the mask and inserting in its place a

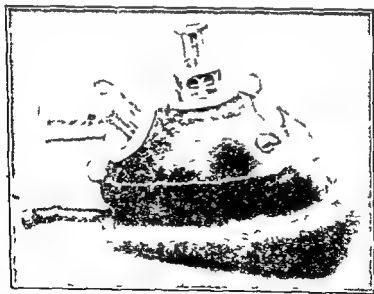


FIG. 11. The Mehta mask (*Brit med J*).

Salivary expiratory valve. This allows an anaesthetist with a small hand to support the jaw and at the same time to close the spring loaded expiratory valve during bag pressure. An attachment to allow a stomach tube to be used with a face mask has been described by

its operation, but a temperature control unit or automatic thermo compensator maintains a constant vapour concentration independent of changes in ambient temperature. This device, a small metal bellows containing a liquid is fitted within the vaporizing chamber below an orifice which is made smaller as the temperature rises. Medical gases can be added to the inhaler without altering the indicated vapour concentration. The concentration control lever, which moves over a scale showing the vapour strength in volumes per cent, the ether level indicator and the ether filler are mounted on the top of the apparatus. The inhaler contains a water compartment which acts as a heat buffer. This is commonly used as a draw over apparatus for the administration of ether vapour and air or a mixture of medical gases can be added to the ether mixture. Should the flow rate of these gases be insufficient air would be drawn through the inlet valve and no resistance to inspiration would occur. It is designed to accompany the Oxford Inflating Bellows. If the inhaler is to be used in tropical countries cold water can be put in the water compartment or cold from ether vaporization can be employed to reduce the temperature of the inhaler.

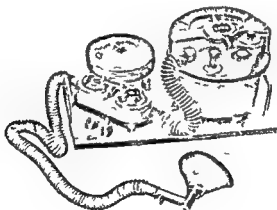


FIG 5 E M O ether inhaler and Oxford inflating bellows
(Pentland Instrument Co Ltd Oxford)

The Oxford Inflating Bellows¹ is designed to be used with the E M O Inhaler and fulfils three main purposes (1) It gives artificial respiration with air or air and additional oxygen (2) It is used to assist or control breathing during anæsthesia (3) It shows the

extent of normal respiration. Rebreathing is prevented by unidirectional valves. The Radcliffe Respiration Pump can be used with it if mechanical inflation is required (see page 48).

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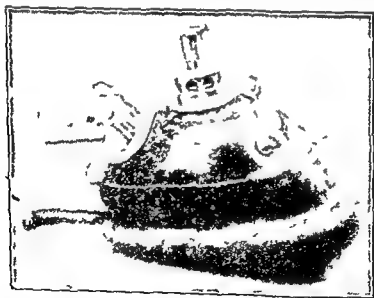


FIG. 6 The Mehta mask (*Brit med J*)

Salt expiratory valve. This allows an anaesthetist with a small hand to support the jaw and at the same time to close the spring loaded expiratory valve during bag pressure. An attachment to allow a stomach tube to be used with a face mask has been described by

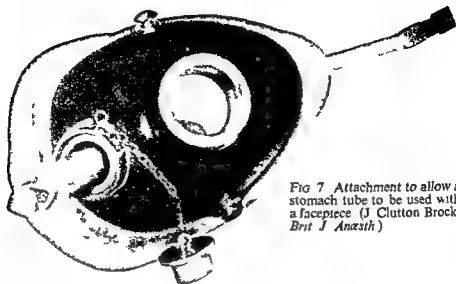


FIG 7 Attachment to allow a stomach tube to be used with a facepiece (J Clutton Brock *Brit J Anæsth*)

Clutton Brock⁴⁶ who has adapted Morton's mask for this purpose See Fig 7

An improved apparatus for the use of Vinesthene has been reported by W J Keating The container will take a 3 or 5 ml vin esthene ampoule unopened or other liquid volatile agent Leakage of volatile anæsthetic does not occur a boon in hot climates By manipulation of a valve drops of anæsthetic can be added to the circuit at a controlled rate The designer of the apparatus recommends vinesthene for use in the Dental and Out Patient Departments See Fig 8

A useful new portable anæsthetic apparatus has been described by J B Stirling and is known as

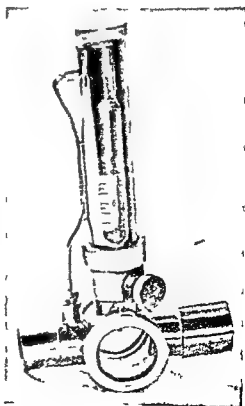


FIG 8 Keating's vinesthene drip (*Anæsthesia*)

the Wright Portable Machine² This is a sensibly robust and easily-portable piece of apparatus. It uses either one 36 gallon oxygen cylinder and two 100-gallon nitrous oxide cylinders, or one 18 gallon oxygen cylinder and two 50-gallon nitrous oxide cylinders. Between

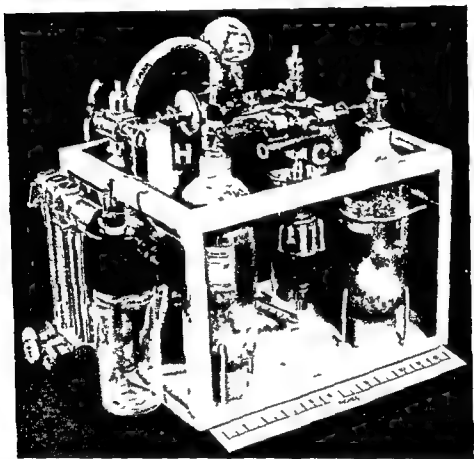


FIG 9 The Wright portable anæsthetic machine. (*Anæsthesia*)

the nitrous oxide cylinders and their Adams valve a 'Klinger' cock is interposed, so that it is unnecessary to turn off one cylinder before the other is turned on. Fresh gas from the new cylinder is instantly delivered if the handle of the cock is rotated through 90°. The empty cylinder can then be replaced at leisure without interrupting the anæsthesia. The apparatus weighs 15½ lb without cylinders and the Klinger cock is obtained from Messrs Small Son & Company of Glasgow.

The Americans have given some attention to the manufacture of a small portable anæsthetic apparatus, and an ingenious little machine has been devised by R A Hingson⁴ This is a portable closed circuit apparatus using midget cylinders similar to sparklet cylinders containing carbon dioxide One loading with cyclopropane and helium mixture and four cylinders of oxygen and helium are sufficient for twenty minutes anæsthesia The apparatus, designed for such purposes as dental extractions and incisions of abscesses under domiciliary conditions etc, weighs less than 1 lb It can be used in association with another small piece of mechanism the size of half an orange which derives its power from a compressed oxygen or nitrogen and oxygen cylinder and provides intermittent positive/negative pressure with either 100 per cent oxygen or 50 per cent oxygen and nitrogen at any desired rate of respiration

Is there an anæsthetist who has not allowed a gas, or worse still, an oxygen cylinder to become exhausted while his attention was elsewhere? This error which has led to deaths, can be prevented by the employment of one of the devices for giving warning of impending failure of a gas or oxygen cylinder^{51 5}

Trilene Vaporizers

When giving trilene to patients in the dental chair, many anæsthetists at present employ a Rowbotham chloroform bottle, but a new device has been described by W J Finnie⁵ This author states that most trilene vaporizers incorporate a glass bottle which can be easily broken and which may leak at the cement metal to glass junction He thinks that most trilene vaporizers are incapable of delivering a strong concentration smoothly and quickly The apparatus is made of cast aluminium and is composed of two tubes each of which will take a 6 ml ampoule of trilene The vaporizer is plugged into the gas exit of an anæsthetic machine Gases can be made to pass directly through the apparatus or through the vaporizing chamber or any intermediate position by movement of a control lever A movement of a knob will fracture the ampoule The majority of patients are managed satisfactorily with the control lever F set at the half mark the more resistant requiring it to be moved to three quarters The second ampoule is held in reserve Laboratory tests have shown that the apparatus causes no marked restriction to breathing while the increase in trilene vapour percentage is smooth and gradual without sudden rises in concentration This vaporizer is made by Cyprane Ltd of Keighley, Yorkshire See Figs 10 and 11

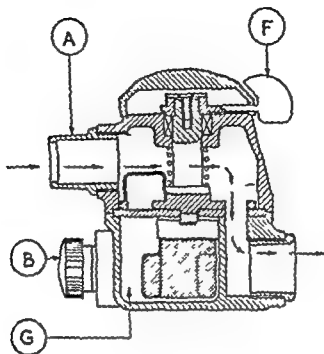


FIG 10 Section of the Finnie trilene vaporizer

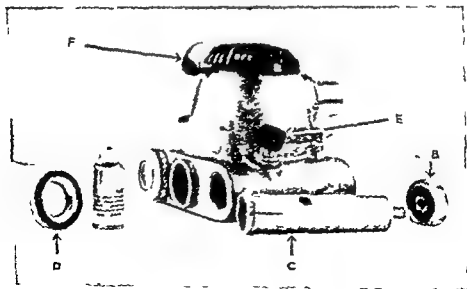


FIG 11 The Finnie trilene vaporizer (*Brit J Anaesth*)

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and air. This weak setting ensures that at minute volumes less than 8 litres a minute the percentage of trilene in air is less than 0.35 per cent. If the minute volume rises above 16 litres per minute 0.4 to 0.5 per cent of trilene vapour is delivered to the patient. The inhaler is designed for analgesia by self administration in obstetrics, etc., and is known as the Burns-Benson Inhaler. See Figs 12 and 13.

The Emotril Trilene Inhaler²⁸ in its modified form has been accepted by the Central Midwives Board for use by midwives working alone. It permits a constant strength of trilene vapour to be inhaled by the patient and thus is uninfluenced by the circumambient temperature (between 60° and 95° F), the respiratory minute volume (between 6-15 litres a minute) and the respiration rate (between 12-30 each minute). The concentration is normally 0.5 per cent in air, but a weaker mixture of 0.35 per cent can be provided. See Fig 14.

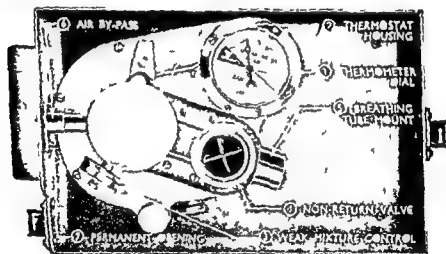


FIG 14 The Emotril (Epstein Macintosh Oxford) trilene inhaler (M I E, Ltd)

The Airlene Inhaler is another portable trilene inhaler made by Airmed Ltd. It incorporates a mixture adjustment collar having three settings labelled 0.35 per cent, 0.5 per cent and 1 per cent trilene vapour in air respectively. The low concentration is suitable for a drowsy patient in labour, the high for producing analgesia or light anaesthesia quickly. A thermocouple control unit compensates for temperature changes, while alteration in tidal volume and respiratory rate do not alter the concentration of vapour. No pure liquid trilene is present when the instrument is in use and over filling or

A constant strength trilene inhaler has also been described⁶ This is designed primarily for the production of a constant percentage of trilene vapour, which is kept between 0.4 and 0.6 per cent for all types of respiration between 7 and 20 litres per minute. It delivers a fixed amount of liquid trilene into every litre of air reaching the patient that is about 0.02 ml per litre. At all likely temperatures

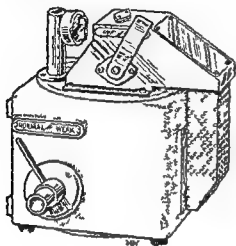


FIG 12 Burns Benson trichloroethylene inhaler (*Brit med J*)

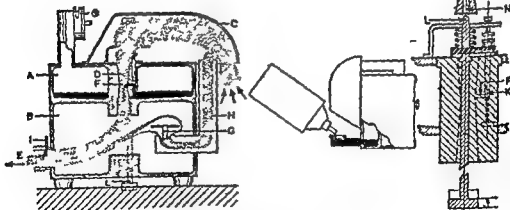


FIG 13 Left Simplified cross section of inhaler
Centre Filling position Right Safety device

this amount will be vaporized so no thermostat is required. A needle valve cut out device prevents alteration in vapour strength consequent on shaking. The inhaler is filled, when turned on its side with 60 ml of trilene. By moving a lever it is possible to weaken the mixture reaching the patient to about 0.35 per cent trilene vapour.

spilling is impossible. One charge is sufficient for several hours' intermittent or thirty minutes' continuous use. This apparatus is also likely to find its chief use in obstetrics. See Fig 15.

The Tecota Mark 6 apparatus has been approved by the Central Midwives Board for use by unsupervised midwives. It is a temperature-compensated inhaler and is manufactured by Cyprane Ltd of Keighley, Yorkshire. It maintains a delivery concentration of 0.5 per cent trilene in air within a wide range of room temperatures (55 to 95° F), tidal volumes and respiratory rates. Obstruction

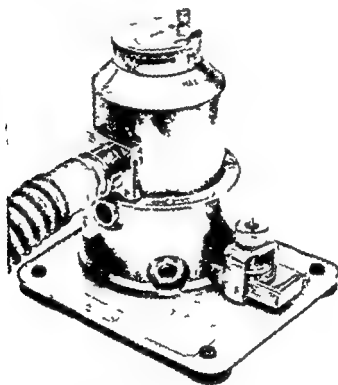


FIG 16 The Tecota Mark 6 trilene inhaler (Cyprane Ltd)

of the air inlet will not result in a stronger mixture being delivered to the patient. Two settings are provided, a maximum delivering 0.5 per cent and a minimum 0.35 per cent trilene vapour in air. Change of position and shaking do not alter its performance. These instruments are checked and sealed by the National Physical Laboratory before being delivered to the user. The weight of the apparatus in its case is 7 lb. See Fig 16.

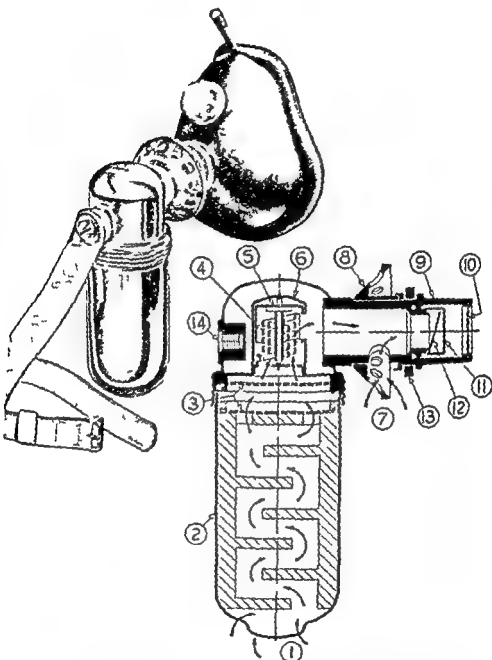


FIG 15 The Airlene trilete inhaler . (Diagrammatic) (Airmid Ltd)

1 Primary air inlets 2 Vaporizing chamber 3 Gauzes
 4 Driving rotor 5 Rotor stator 7 Secondary air inlets
 8 Mixture adjustment collar 9 Tapered fitting for facepiece or rubber
 hose 10 Gauze 11 Non return valve cage 12 Non return valve
 13 Locking ring 14 Screwed hole for attaching clamp bracket or
 wrist strap



FIG 19 The Blease non return valve for use with resuscitator (*Anæsthesia*)

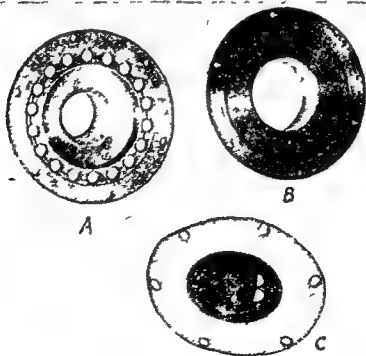


FIG 20 The Blease non return valve casing and diaphragm (*Anæsthesia*)

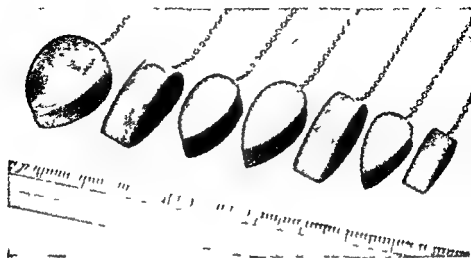


FIG 17 The Blair-Gould dental prop (*Anæsthesia*)

An improved dental prop has been described²⁰ This can be used instead of the traumatic Mason Gag for changing sides. It is made by Medical Pneumatics Ltd of solid rubber. See Fig 17

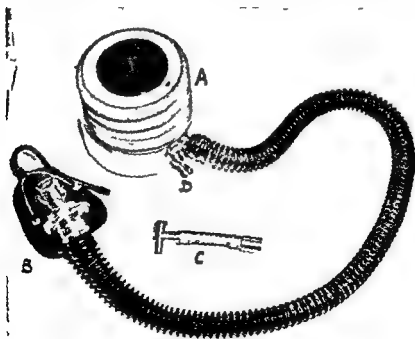


FIG 18 The Blease Manual resuscitator (*Anæsthesia*)

inflate the lungs of apnoeic patients is 20 to 25 cm of water, and should the chest be open 10 to 15 cm of water are sufficient, but this must again be increased to nearly the former figure if packs and retractors confine the lung. An artificial respiration machine should provide for a phase of rapid inflation with short peak pressure followed by a prolonged and complete expiratory phase at atmospheric pressure. This arrangement avoids undue embarrassment to the filling of the right heart and to the circulation through the lung. It allows for fairly complete expulsion of carbon dioxide. Another type of automatic machine produces, in addition to positive pressure inhalation negative pressure exhalation. The desirable criteria by which such a machine should be judged are as follows —

It should reproduce as nearly as possible the normal gas flow and volume (normal pressure changes of course can never be reproduced). It should be sensitive to increase and decrease of intra thoracic pressure so that the change from inspiration to expiration occurs at a pre set pressure. It should enable the anaesthetist to detect leaks, obstruction and mechanical faults. It should be quiet in operation, robust and mechanically reliable and should not increase the risk of explosion when oxygen and flammable gases are in use.

Automatic respirators should only be used by skilful anaesthetists if manual inflation is not for one reason or another, available. Like all machines they require an intelligent human brain to control, oversee and if necessary take over their operation, while the anaesthetist deprived of the information which long experience of the feel of the bag gives, is deprived of a real sixth sense in the conduct of the anaesthesia or the ventilation. Respirators may be pump-controlled which requires a safety valve to prevent the build up of excessive pressure. They may on the other hand be pressure sensitive when the change from one phase of respiration to the other takes place at a predetermined pressure.

Pump type respirators include machines devised by James (1950)¹² and Beaver (1953)¹³. Pressure sensitive respirators include the Vivrator the essential part of the Spiropulsator driven by compressed air; the Pulmopulsator, a German machine worked by a compressor; the Blease Pulmoflator, also worked by a compressor; the Williams Respirator¹⁴ which is designed to work from the anaesthetic gases of the Boyle machine on a semi-closed circuit, and the Aintree Respirator¹⁵. Pressure sensitive respirators using electricity instead of compressed gas to actuate the mechanism include the Bang Respirator¹⁶ the Newcastle Respirator,¹⁷ the Clevedon Respirator,¹⁸ the Morsch Respirator¹⁹

Machines for Artificial Respiration

Much ingenuity has been expended in recent years in the design and manufacture of artificial respiration machines both those operated by hand and those operated mechanically. The Blease Manual Resuscitator⁷ consists of a Connell type bag connected to a non return valve and face mask or endotracheal tube. A tap allows oxygen to be added to the bag. The special feature is the non return valve which has no resistance to expiration because of the negative pressure which develops in the concertina bag during ventilation. The inflation pressure is normally less than 15 mm of mercury and the apparatus is described as being a highly efficient one which can be used for ECT without additional oxygen and for artificial respiration. Another simple respirator⁸ employs the ingenious idea of utilizing a Tex suction operated motor wind screen wiper and a concertina bag removed from a Coxeter Mushin absorber. It is worked off a source of suction which is capable of giving a negative pressure of 20 mm of mercury and will even work off a water tap and is thus independent of the electricity supply.

This interesting and highly topical question has been well reviewed by W W Mushin and L Rendell Baker¹¹. They point out that while efficient machines were made by Janeway and others in the second decade of the present century it was the work of Freckner and Crafoord of Stockholm who in 1934 described their *spiro-pulsator* which created interest in automatic respirators. The substitution of manual inflation for the tank respirator by Lassen and Ibsen¹² during the poliomyelitis epidemic in Copenhagen in 1953 created the need for such instruments on a large scale to take the place of an ever necessary pair of hands. The pressure required to



FIG 21 The Mortimer respirator
Operated by suction windscreen
wiper

inflate the lungs of apnoeic patients is 20 to 25 cm of water, and should the chest be open 10 to 15 cm of water are sufficient, but this must again be increased to nearly the former figure if packs and retractors confine the lung. An artificial respiration machine should provide for a phase of rapid inflation with short peak pressure, followed by a prolonged and complete expiratory phase at atmospheric pressure. This arrangement avoids undue embarrassment to the filling of the right heart and to the circulation through the lung. It allows for fairly complete expulsion of carbon dioxide. Another type of automatic machine produces in addition to positive pressure inhalation, negative pressure exhalation. The desirable criteria by which such a machine should be judged are as follows —

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The Fazakerley Respirator² is designed to provide anæsthetists with a means of controlling both inhalation and exhalation, and of assisting the patient in exhaling. It gives positive inflation pressures of 10–30 cm. of water and provides for control of the frequency and the phases of the respiratory cycle. It can be made to develop a negative pressure in the patient's airway during his expiratory phase, and is run by compressed air 20–30 litres a minute at 100–360 mm. of mercury pressure.

Two more respirators have recently been described by the workers of the Newcastle school.⁴⁴ Both are of the intermittent positive pressure type, while in both, the lungs are inflated to a predetermined pressure. They are designed to give rapid inflation and prolonged expiration and a pause, so that the mean intrathoracic pressure can be kept low. When suction is available (e.g. from a vacuum cleaner motor or an oxygen injector device) a negative pressure can be applied to the lungs during expiration. The machines are pneumatically operated from low pressure sources of gas or air at pressures between 30 and 70 mm. of mercury. Rates of respiration are controllable between 4 and 50 per minute, and by the addition of a triggering valve respiration can be made sensitive to the efforts of the patient.

The Radcliffe Respiration Pump⁵ (H. G. East and Co., 37a Oxford Rd. Cowley Oxford) is designed to provide intermittent positive pressure respiration (I.P.P.R.) through a cuffed tracheotomy tube for treating respiratory inefficiency in poliomyelitis⁶ and in conditions such as polyneuritis and tetanus during curarization. The new type which has proved to be most reliable and efficient, is provided with a negative pressure phase of 0 to minus 10 cm. of water. It can be operated either from the mains or from a 12-volt battery.

Dental Apparatus

Changes in anæsthesia for dental extractions have been concerned more with change of agent than with change of apparatus. Cyclopropane has invaded the dental office but as yet has attained no very wide popularity. Gas and oxygen remains above all the most popular dental anæsthetic. A new nasal inhaler²¹ combines the advantages of the Goldman and Karn inhalers. It is provided with a special type expiratory valve and an on and-off valve with an air inlet. The inhaler is attached to the face with Connell's harness (Medical Pneumatics Ltd.)

J G Bourne has designed a small, light and ingenious piece of apparatus for the administration of cyclopropane in the dental chair⁴⁸ Two standard Sparklet bulbs, distinctively coloured, are bound together in pairs with transparent adhesive tape. They contain cyclopropane and oxygen nitrogen mixture, respectively, in amounts which, when discharged into the 6 litre bag fill it in the proportions

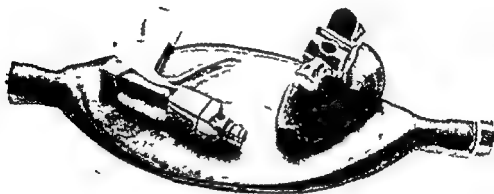


FIG 22 Bourne's apparatus for the administration of cyclopropane in the dental chair (*Sparklets Ltd*)

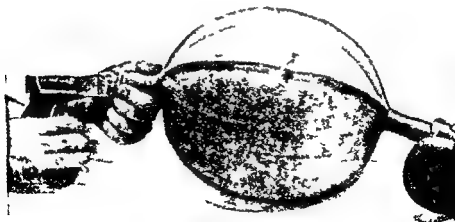


FIG 23 Bourne's apparatus ready for use (*Sparklets Ltd*)

cyclopropane 50 per cent oxygen 25 per cent and nitrogen 25 per cent. The trigger of the 'pistol' is a lever which presses the bulbs on to their piercing pins. For filling, the muzzle is momentarily plugged into the bottom of the bag which incorporates a non return valve. The mask has been specially designed to fit the face of an adult when the jaws are propped wide open. It is connected to the bag by an angle piece with a two way stopcock. In one position of the stopcock the bag is shut off and the patient is able to breathe air; in the other position, the air vent is closed and the patient breathes in and out of the bag. (See also Chapter 2.)

Since the mixture in the bag is outside the limits of ignitability, anti static rubber is not necessary. It must not be forgotten however that if the contents of the bag are discharged into the air the cyclopropane would become diluted and therefore ignitable though not, in the usual sense explosive. See Figs 22 and 23.

Expiratory Valves

Many anæsthetists have been faced with the problem of opening or closing the expiratory valve situated near the patient whose head has been completely wrapped up in sterile drapes. This problem has been solved in the Cardiff Remote Control Respiratory Valve⁹. This is an improvement on the apparatus described in 1950¹⁰. The "shut off" mechanism is actuated by remote control via a Bowden cable and is separate from the very lightly sprung expiratory valve disc which operates during spontaneous respiration. It is manufactured by MIE Ltd of New Cavendish Street, London, W1. See Figs 24 and 25.

Much new work has been done on the physics and applied engineering of semi closed circuits and on the characteristics of expiratory valves. W W Mushin and W W Mapleson³ in an interesting article dealing with the pressure flow rate characteristics of expiratory valves commence with the assumption that the setting of the expiratory valve determines the mean pressure in the anæsthetic circuit and the patient's respiratory tract which in turn determines the effort required to expire through the valve. It also determines the size of the physiological dead space because as the mean pressure in the respiratory tract is raised the functional residual air and hence the dead space becomes increased. In addition when partial rebreathing is employed the opening pressure of the expiratory valve must be slightly greater than the collapsing pressure of the reservoir bag or the bag will not act as a reservoir. In the case of a one gallon bag the opening pressure of the valve must not

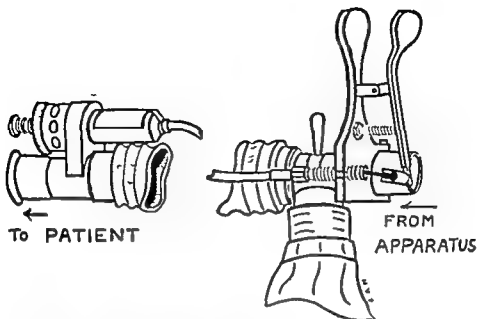


FIG 24 The Cardiff remote-control expiratory valve (*Brit J Anaesth*)

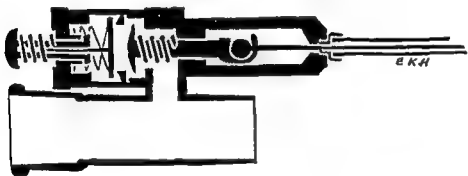


FIG 25 Cardiff remote-control valve in section (*Brit J Anaesth*)

be less than 0.5 to 1 cm of water. They emphasize that the setting of the valve is not the main method which governs rebreathing, the extent of which is more dependent on the relationship between the minute volume respiration of the patient and the flow of fresh gases into the circuit. They conclude that the ideal valve would open in the inverted position at a pressure of 0.5 cm of water. In the upright position of the valve this pressure should be exceeded as little as possible. In clinical practice an adjustable compression spring is unnecessary if the patient is breathing as the valve should

always be at minimal pressure. Many workers, however, find an adjustable valve useful when employing intermittent positive pressure respiration with a so-called controlled leak.

Carbon Dioxide Elimination in Anæsthetic Circuits

The problem of the removal of carbon dioxide from anæsthetic circuits has recently received much attention from investigators.⁵⁰ Removal of carbon dioxide consists of the reduction of the percentage of the gas from about 4 per cent to approximately 0.03 per cent or 0.04 per cent. Soda lime is 4 per cent sodium hydroxide, 70 per cent calcium hydroxide, 17 per cent water with more or less calcium carbonate as a binder. It is in physical terms a 20 per cent aqueous solution of sodium hydroxide dispersed on calcium hydroxide.

Four methods of carbon dioxide analysis are commonly used: they are infra red analysis, differential absorption, mass spectrometry and photometry with an indicator. The Brinkman Carbovisor belongs to the last group. It is generally assumed that arterial and alveolar carbon dioxide tensions (end expiratory samples) correspond in health although disease may alter this relationship.

It would appear that carbon dioxide rebreathing is best prevented⁵⁰ by the use of a unidirectional circle type absorber with soda lime as absorbent in a semi-closed system and with a total gas flow of not less than 7 litres a minute in adults, with the expiratory valve as near to the patient's lips as possible. The unidirectional circle type of absorber is superior to the to and fro Waters type of absorber as an eliminator of carbon dioxide mainly because of rebreathing from the dead space of the latter. There is some evidence that the unidirectional circle absorber is more efficient than the to and fro circle type⁵⁰ (e.g. the Coxeter-Mushin).

There is no doubt that many of our anæsthetized patients suffer from hypercapnia which is a physiological trespass. On the other hand hyperventilation occasionally causes a respiratory alkalosis (which is usually harmless) and may cause some hypotension. In addition it may give rise to a diminution of blood supply to the brain from cerebral vasoconstriction and a stagnant tissue anoxia due to peripheral vasoconstriction shown by a lilac colour of the skin (Scurr).²³ There may be in addition a shift of the oxygen dissociation curve to the left. In spite of these results of overventilation the general consensus of opinion is that it is better to err on the side of over- than of under-ventilation.

The elimination of rebreathing in various semi-closed systems²⁴ has been studied and has extended the theoretical analysis which

Molyneux and Pask²⁵ applied to the Magill rebreathing attachment (A) to other types of semi-closed system. Five systems were considered. From this work it is concluded that the classical Magill's

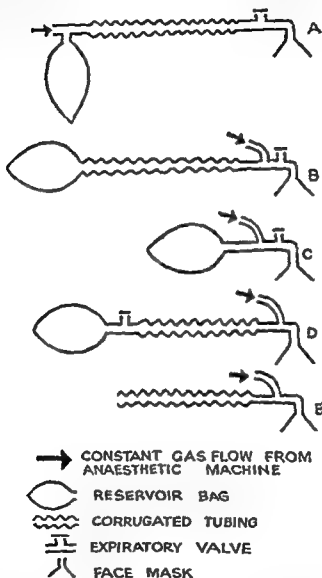


FIG. 26. Diagrams of semi-closed circuits (Mapleson). (*Brit J Anaesth*)

attachment has in so far as carbon dioxide elimination is concerned a marked advantage over the others regarding economy of gases, and rebreathing with this attachment has probably been eliminated if the flow of fresh gases is at least equal to the patient's minute

volume. System E has the least resistance to breathing and is the only one dispensing with an expiratory valve. In the other systems (B, C, and D) the flow of fresh gases must be about twice the minute volume of the patient. See Fig. 26.

Further experimental work on rebreathing with a semi-closed circuit has been described by Woolmer and Lind,³ who have made a laboratory investigation into the completeness or otherwise of

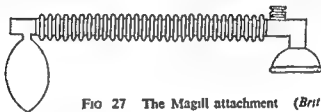


FIG. 27 The Magill attachment (*Brit J Anæsth*)

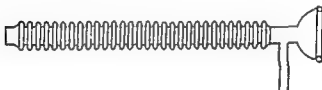


FIG. 28 The T piece arrangement (*Brit J Anæsth*)

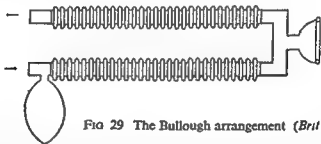


FIG. 29 The Bullough arrangement (*Brit J Anæsth*)

carbon dioxide elimination during anæsthesia with a semi-closed circuit. Three arrangements were tested. In the first the corrugated tubing, reservoir bag and expiratory valve known as the Magill attachment, were used. In the second the T piece attachment* with neither valves nor bag but with rebreathing tube. In the third the arrangement described by Bullough²¹ which combines elements of both the above systems and consists of a reservoir bag and corrugated

* The T piece attachments used in these experiments and the carbon dioxide elimination resulting from their use are actually very different from those originally described by Philip Ayre²² in which the internal diameter of the reservoir tube should be 1 cm. so that each inch in length has a capacity of 2 ml.²³

tube into which the fresh gases flow as with Magill attachments, instead of an inspiratory valve near the facepiece, a second open ended corrugated tube carries excess gas flow away from the patient. The conclusions reached were as follows. A flow rate of 7 litres a minute into a Magill attachment limits carbon dioxide rebreathing to a satisfactory level in an average adult patient. The tension of the expiratory valve does not influence elimination of carbon dioxide although a tight valve increases dead space and forms an obstruction to expiration. Magill's attachment is superior to the T-piece system for carbon dioxide elimination.

These results agree fairly well with those obtained by another group of investigators⁴ who in a clinical analysis of carbon dioxide elimination from semi closed systems, show that in adults the Mapleson E (T piece) and Mapleson B systems (Fig 26) both return a high concentration of carbon dioxide to the patient, and are both inferior in this respect to the Mapleson A system, the well known Magill attachment.

The carbon dioxide concentration in Boyle's machine has been considered by Harrison and Jones⁶. They gave oxygen at the rate of 8 litres a minute with a Magill attachment, and found that at the proximal end of the corrugated tube a mean of 0.53 volumes per cent of carbon dioxide was present. This is 17 times the volume of room air, which is 0.03-0.04 volumes per cent.

A technique has been reported by Bullough⁷ for converting standard British apparatus for use with a non rebreathing technique. He rightly emphasizes that a non rebreathing technique has the following advantages: (1) The expired gases are not inhaled. (2) The inhaled mixture is identical with that delivered from the machine and so contains no nitrogen, water vapour or carbon dioxide. (3) The carbon dioxide concentration is not artificially raised. (4) Nitrogen elimination is more rapid. Two methods are described using the Boyle-Coxeter-Mushin or the Boyle Mark 2 absorber. In the first the circle absorber is used as a semi closed circuit with the soda lime canister turned off. The expiratory valve at the facepiece is open and a flow of gases of 8 litres a minute is set going. A clamp is now placed on the expiratory hosing near the facepiece. In the second method the twin corrugated tubes from the absorber are removed and the expiratory port is blocked with a cork or rubber cap from a standard saline transfusion bottle. The Magill attachment is plugged in to the inspiratory port and the reservoir bag control is turned to shut. The expiratory valve near the facepiece is opened and a flow of 8 litres a minute of fresh gases provided.

The unidirectional valve at the inspiratory port acts as a gravity check valve. There must be sufficient tension on the expiratory valve to allow the reservoir bag (of the absorber circuit not the Magill attachment) to fill if the distal end of the tube is occluded. A supply of fresh gases at least equal to the patient's minute respiratory volume must be used. If the flow is less than the patient's minute respiratory volume the bag will empty so a rough estimate of the patient's respiratory minute volume can be rapidly performed. This is useful at the end of an operation in which muscle relaxants have been employed.

An ingenious non rebreathing valve has been described⁸ in which the expiratory port automatically closes when the reservoir bag is compressed in assisted or controlled breathing. It is made of Perspex and has a dead space of 9 ml and a resistance of 1 cm of water during expiration. A non return valve is useful also during or at the end of an operation as the efficiency of spontaneous respiration can be measured in the following way. The total gas-flow from the flow meter is set so that the bag keeps a constant average size at the end of expiration. A decrease in bag size indicates that the respiratory minute volume is greater than the gas flow. An increase in bag size shows that the R M V is less than

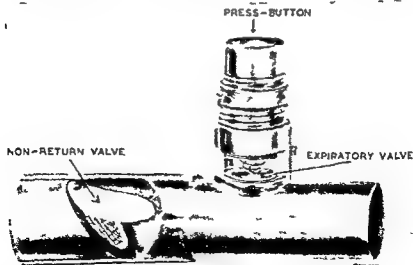
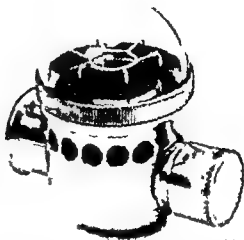


FIG 30 The Ryan non rebreathing valve (*Brit J Anaesth.*)

FIG. 31 A non return valve (A J H Hewer)
(Airmed Ltd)



the gas flow. A useful non rebreathing circuit can be made if a one-way valve is incorporated in the circuit preferably near the expiratory valve^{30, 37} See Figs 30 and 31

The chemical control of soda lime efficiency in certain absorbers has been investigated by Pires.³⁸ Many authors have shown that the signs of carbon dioxide retention become less evident as the depth of anaesthesia increases. It is probably innocuous in concentrations less than 0.5 per cent in spite of the fact that the normal content of air is 0.04 per cent but increasing concentrations above this figure are undesirable. The Draper carbon dioxide detector which is a simple accessory consisting of two ball valves, two bubble tubes equipped with tightly fitting rubber connections and a rubber bulb mounted in series with the inspiratory side of the circle absorber is a useful accessory. By squeezing the bulb its negative pressure draws a sample of mixture about to be inhaled and allows it to bubble into the test tubes. The first test tube contains water to trap any soda lime trace that may be present. The second contains the chemical reagent a solution of calcium hydrate 80 g per litre. This is first calibrated with a known carbon dioxide oxygen mixture. Thereafter the number of squeezes of the bulb which are required to produce a certain standard of cloudiness gives an accurate indication of the amount of carbon dioxide present in the circuit.

The measurement of controlled respiration has been investigated by Eastwood and Harbord³⁹ who describe an apparatus which can be manually operated for artificial respiration. It draws a consistent tidal volume of gas mixture from a spirometer, passes it through a

Waters canister and returns it to the spirometer for measurement. Movements of the spirometer are recorded on a revolving drum and the gas flow is regulated so that the record remains horizontal. The circuit must, of course, be gas tight. This has been modified³¹ to allow for its use with nitrous oxide and oxygen without carbon dioxide absorption the whole of each expiration being removed. The machine is manufactured by Messrs Cypranc Ltd, of Keighley.

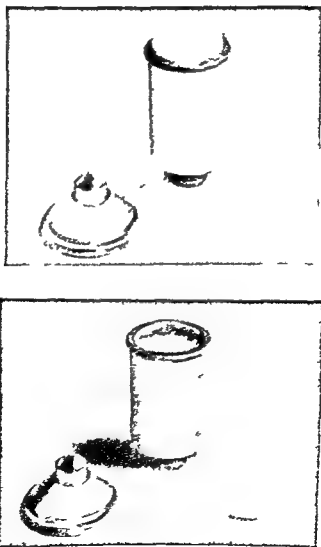


FIG 32. Waters canister with potscrub (Robson and Pask) (*Brit J Anaesth*)

In spite of the multiplicity of complicated circle absorbers, many anaesthetists remain faithful to the Waters canister, which is inexpensive, portable and easily sterilized. The performance of this canister has been investigated afresh²⁵ and it has been found that the Waters canister absorbs carbon dioxide most efficiently if, after the soda lime has been shaken down as tightly as possible, a simple nylon pot scraper is inserted so as to leave about half of it to be compressed by the wire gauze in the lid of the canister when the cap is screwed on. This prevents channelling of the gases and makes for efficiency with the canister lying on its side.

Soda Lime Continuing research has led to the commercial introduction of Durasorb, a new soda lime with prolonged effective life which does not overheat. While it contains no bonding material it is relatively free from dust. The size of the granules is 4-8 mesh and their colour is pink when active and white when inactive.

ANÆSTHESIA APPARATUS FOR INFANTS AND CHILDREN

H B C Sandiford²⁷ has described an apparatus designed to reduce dead space which is of prime importance in a small patient whose tidal volume may be as little as 16 to 20 ml. It consists of a new type of mask and valve with resistance of 5 mm of water with a small bag. The dead space is about 5 ml. The mask fits well up to about 4½ years of age. In addition, a small to and fro absorber is described in which too dead space has been reduced to a minimum.

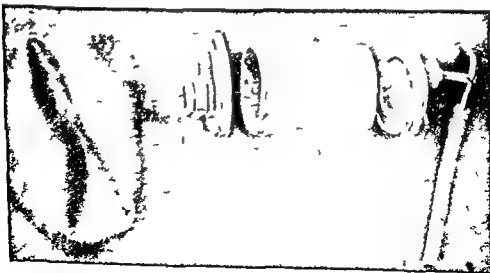


FIG 33 The Sandiford to and fro absorber for children (*Anæsthesia*)



FIG 34 The Sandiford semi-closed children's inhaler (*Anæsthesia*)

The ports are placed eccentrically to prevent channelling down the side of the canister. An endotracheal tube is mounted on a T-piece which is bent to avoid the need for an angle piece. The fresh gases enter from the bag through the absorber and if the rubber tube or the outlet of the T piece is clamped the absorber is automatically brought into use. Thus a semi open circuit can rapidly be converted to a closed one which enables assisted respiration to be carried out.

Gas Analysis

The methods of gas analysis have been reviewed by R. Woolmer³⁸ who in a masterly description of this to the average anæsthetist complicated subject points out that gas analysis is becoming increasingly important in anæsthesia and divides the possible methods into chemical and physical. The former is apt to be slow so that changes occurring during the progress of an anæsthetic cannot be measured until afterwards and so cannot influence the minute to minute control. Physical methods which have been used include estimation of molecular weight, frequency at which electro magnetic radiation is emitted or absorbed, relationship of ionic loss to ionic charge, heat conductivity, velocity of transmission of sound, magnetic susceptibility, refractive index and viscosity. These various methods are described, although fully to understand the principles on which they are based requires a knowledge of physics which few anæsthetists possess.

A portable oxygen analyser³⁹ is described in which a test atmosphere is drawn into the instrument by aspiration from a rubber bulb and is made to displace the gas mixture originally present in the

machine. On depressing a switch a spot of light on a scale indicates the percentage of oxygen present. It covers a range from 0 to 100 per cent with an accuracy of ± 2 per cent. It depends on the Pauling principle which utilizes the fact that oxygen is the only gas commonly used in anaesthesia whose molecules are attracted by a magnetic field. Nitric oxide and chlorine dioxide share this paramagnetic property with oxygen. Light is supplied from two small dry batteries which together with the drying agent, self-indicating silica gel, are the only replaceable components.

A modification of Exton's method for blood oxygen estimation has been described.⁴⁰ This method increases the speed and convenience of estimating the oxygen content of blood originally described by Exton and his colleagues in 1945.⁴¹

ANTISEPSIS OF ENDOTRACHEAL TUBES AND MASKS

There is always a danger that infection may be transferred from one patient to another by means of anaesthetic face masks and endotracheal tubes. It appears too that soda lime is not an effective barrier to the passage of micro organisms. Autoclaving has deleterious effects on rubber and the application of an antiseptic requires most careful after-cleaning to prevent irritation of the patient's tissues. There is evidence that washing in a preparation containing 3 per cent hexachlorophene, a highly bacteriostatic substance which is stable in bar and liquid soap, non-allergic and non-irritating to tissues, removes most pathogenic organisms. Scrubbing for 1 minute inside and outside is recommended.⁴²

Electronic Aids

A Transistor Pulse Counter⁴⁴ This ingenious piece of apparatus continuously displays the pulse rate on a dial with a needle which flicks, while a small loudspeaker which emits a sound each time the heart beats is also provided.

The Stanco Oximeter (Stanley Cox Ltd, 11 Gerrard Street London W 1) This is an instrument for measuring arterial oxygenation in the human without recourse to arterial sampling and chemical analysis. With it it is rapidly possible to read the patient's arterial oxygen saturation and to note any changes taking place. Its operation is simple and while setting up procedures take about one minute, subsequent readings require only a few seconds to make. It weighs 15 lb and is portable. The capillaries of the ear lobe are first arterialized with a vaso-dilator histamine containing cream and a small plastic earpiece is applied to the ear. The light from a



FIG 35 The Stanco oximeter earpiece correctly attached
(Stanley Cox Ltd)

tungsten filament lamp contained in the plastic earpiece is transmitted from the ear and the outputs from red and infra red responsive cells are compared. Among its many and varied applications are the following. The checking of the efficiency of respiratory aids, the study of cardiac function, the observation of the arterial oxygen saturation level during anaesthesia and in the post operative period.

References

- 1 *Brit med J* 1953 2 202 (July 25)
- 2 KEATING W J 1953 *Anaesthesia* 8 197 (July)
- 3 STIRLING J H 1955 *Anaesthesia* 10 308 (July)
- 4 HINGSON R. A. 1954 *J Amer med Ass* 156 604 (Oct 9)
- 5 FINNIE, W J 1954 *Brit J Anaesth* 26 48 (Jan)
- 6 BURNS T H S 1954 *Brit med J* 1 329 (Feb 6)
- 7 KILPATRICK, A. 1954 *Anaesthesia* 9 303 (Oct)
- 8 MORTIMER P L F 1954 *Anaesthesia* 9 312 (Oct)
- 9 HILLARD E. K. 1953 *Brit J Anaesth* 25 268 (July)
- 10 MUSHIN W W and EZARD 1950 *Brit J Anaesth* 22 186
- 11 MUSHIN W W and RENDELL BAKER L. 1954 *Brit J Anaesth* 26 131 (March)

- 12 JAMES N H 1950 *Med J Aust* 1 325
- 13 BEAVER, R A 1953 *Lancet* 1 977
- 14 WILLIAMS T M 1952 *Brit J Anaesth* 24 222
- 15 ESPLEN J R 1952, *Brit J Anaesth* 24 303
- 16 BANG C 1953 *Lancet* 1, 723
- 17 PASK E A 1953 *Lancet* 1 141
- 18 MACRAE J *et al* 1953, *Lancet* 2 971
- 19 MORCH E T 1947 *Proc R Soc Med* 40 603
- 20 BLAIR GOULD R 1955 *Anaesthesia* 10 310 (July)
- 21 BLAIR GOULD R 1954 *Anaesthesia* 9 44 (Jan)
- 22 SCURR, C F 1956 *Proc R Soc Med* 49 229 (April)
- 23 MUSHIN, W W and MAPLESON W W 1954 *Brit J Anaesth* 26 3 (Jan)
- 24 MAPLESON W W 1954 *Brit J Anaesth* 26 323 (Sept)
- 25 MOLYNEUX L and PASK E A 1951 *Brit J Anaesth* 23 81
- 26 HARRISON G G and JONES C S 1955 *Brit J Anaesth* 27 162 (April)
- 27 BULLOUGH J 1955 *Brit J Anaesth* 27 181 (April)
- 28 RUBEN H 1955 *Anesthesiology* 16 643 (July)
- 29 PIRES F K 1953 *Brit J Anaesth* 25 43 (Jan)
- 30 EASTWOOD A B, and HARBORD R P 1954 *Brit J Anaesth* 26 197 (May)
- 31 HARBORD M R 1955 *Brit J Anaesth* 27 146 (March)
- 32 WOOLMER R and LIND B 1954 *Brit J Anaesth* 26 316 (Sept)
- 33 AYRE P 1937 *Lancet* 1 561
- 34 BULLOUGH J 1952 *Brit med J* 1 28
- 35 ROBSON J G and PASK E A 1954 *Brit J Anaesth* 26 333 (Sept)
- 36 RYAN A R 1955 *Brit J Anaesth* 27 102 (Feb)
- 37 SANDIFORD H B C 1953 *Anaesthesia* 8 122 (April)
- 38 WOOLMER R 1953 *Brit J Anaesth* 25 315 (Oct)
- 39 BRACKEN A 1953 *Brit J Anaesth* 25 383 (Oct)
- 40 PASK E A 1953 *Brit J Anaesth* 25 297 (Oct)
- 41 EXTON J *et al* 1945 *J Lab clin Med*, 30 84
- 42 DAVIES R M, VERNER I R and BRACKEN, A 1956 *Brit J Anaesth* 28 196 (May)
- 43 McDONALD, W L J *et al* 1955 *Anesthesiology* 16 206 (March)
- 44 MOLYNEUX L and PASK E A 1955 *Brit J Anaesth* 27 261 (May)
- 45 MEHTA P M 1955 *Brit med J* 1 727 (March 19)
- 46 CLUTTON BROCK J 1953 *Brit J Anaesth* 25 387 (Oct)
- 47 EPSTEIN H G and MACINTOSH R R 1956 *Anaesthesia* 11 83 (Jan)
- 48 BRITISH STANDARDS INSTITUTION 1955 *Anaesthesia* 10 407 (Oct)
- 49 BOURNE J G personal communication (in the press)
- 50 BRACKEN A 1956 *Proc R Soc Med* 49 215 (April)
- 51 ESPLEN J R 1956 *Brit J Anaesth* 28 226 (May)
- 52 HILL, E FALKNER 1956 *Brit J Anaesth* 28 228
- 53 ESPLEN J R 1956 *Brit J Anaesth* 28 176
- 54 HORTON J A G, INKSTER J S, PASK E A 1956 *Brit J Anaesth* 28 169
- 55 RITCHIE RUSSELL, W, SCHUSTER E, CRAMPTON SMITH A, SPALDING J M K 1956 *Lancet* 1 539 (April 28)
- 56 LASSEN H C A 1953 *Lancet* 1 37 (Jan 3)
- 57 HEWER A J H personal communication
- 58 EPSTEIN H G and MACINTOSH R R. 1949 *Brit med J* 1092 (Nov 12)
- 59 AYRE P 1956 *Brit J Anaesth* 28 521 (Nov)

CHAPTER 4

DEVELOPMENTS IN ENDOTRACHEAL ANÆSTHESIA

MANY so called recent advances in endotracheal anæsthesia were in fact made many years ago and to put these into perspective it may be helpful to give a very brief resume of the history of the method

Historical Intubation of the trachea in animals was, so far as is known first performed and recorded by Vesalius¹ in 1542 Snow gave chloroform vapour through a tracheotomy tube to animals in 1858 It is interesting to observe that he used a closed-circuit with caustic potash to absorb the carbon dioxide Trendelenburg applied Snow's method to man in 1871² while MacEwen in 1880 was the first to administer an anæsthetic (chloroform) through a metal tracheal tube introduced through the mouth³ Kuhn and Elsberg⁵ both used endotracheal anæsthesia in suitable cases between 1900 and 1910 while Magill and Rowbotham developed the technique to deal with plastic facial surgery occasioned by the 1914-18 European war⁶ Up to this point the insufflation technique had been used i.e. the anæsthetic gases were forced through a tube whose distal extremity lay in the trachea the return flow either taking a natural course around the tube or an artificial one through a second tube lying in the trachea The insufflation method has now

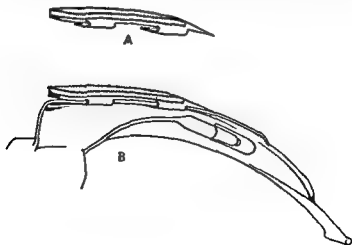


FIG 36 Rubber tooth protector shown A alone B clipped to back of laryngoscope (*Waddy Brit med J*)

been replaced by the inhalation one in which respiration takes place through one wide-bore tracheal tube which may be passed through the nose mouth or through laryngotomy or tracheotomy wounds as required. Any type of open, semi-closed or closed-circuit technique may be employed.



FIG 37 Causes of respiratory obstruction during endotracheal anaesthesia (Ballantine and Jackson *Anaesthesia*)

Errors in technique The methods of performing nasal and oral intubation are now well known and will not be described here. In difficult cases damage is sometimes caused to upper teeth and various methods of protection have been devised. One neat fitting is a rubber guard which can either be attached to the laryngoscope blade by a metal clip⁷ or simply stuck on with an adhesive such as Bostik.⁸

One of the objects of endotracheal anæsthesia is the maintenance of a perfect airway but this may not be achieved owing to a variety of technical errors. The commonest of these are illustrated (Fig. 37) —⁹

(a) Kinking of tubing of catheter mount

(b) Patient is biting on the endotracheal tube in the absence of a prop or airway

(c) Kinking of plain rubber Magill's tube owing to extreme flexion of head

(d) Same fault as in (c) even when Bourne's sheath is in use¹⁰

(e) Narrowing of portex tube softened by warmth

(f) Unguarded end of flexo metallic tube has not been shortened enough and has kinked

(g) Tip of bevel is kinked against trachea

(h) Bevel lies against tracheal wall

The last six faults can be avoided by using a suitably shortened unbevelled flexo metallic tube as shown

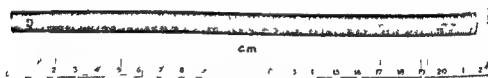


FIG. 38 Unbevelled flexo metallic endotracheal tube (Ballantine and Jackson *Anæsthesia*)

In addition to the mechanical causes of respiratory obstruction already described secretions may collect in the tracheal tube and in the bronchial tree. Large quantities cause bubbling which is easily audible but the smallest amount can be detected by a stethoscope connected to a metal cuff surrounding the connecting tube¹¹. The fluid can then be removed by suction.

A rare cause of obstruction has recently been described³ in which a badly made flexo metallic endotracheal tube developed a blister on its inner surface after prolonged administration of a nitrous

oxide-oxygen-trichlorethylene mixture On further investigation it has been shown that ether vapour can also cause this phenomenon

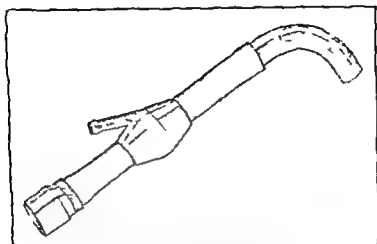


FIG 39 Auscultation cuff for detecting tracheal and bronchial secretions (Laycock *Brit med J*)

Oral intubation by transillumination If the light of the laryngoscope should fail the glottis can usually be seen if a small and intense source of illumination such as a pen torch, is directed on to the skin over the crico-thyroid membrane³⁰ In cases of emergency even the laryngoscope can be dispensed with and homely articles such as a shoe horn or tongue depressor can be substituted It is

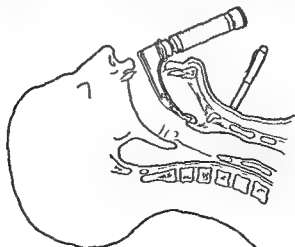


FIG 40 Direct vision laryngoscopy by transillumination (Tate *Lancet*)

worth noting that this method of transillumination was in use in 1859 sunlight being concentrated by lenses and concave mirrors ²¹

Cuffed endotracheal tubes Cuffed tubes are popular if a closed circuit technique is being used or if there is any risk of fluid aspiration. They probably give rise to less soreness than the alternative packing with vaseline gauze. Nevertheless it is important to test the cuff before passing the tube and to inflate with the minimal pressure to ensure a gas tight fit. Cases have been described where tracheal and

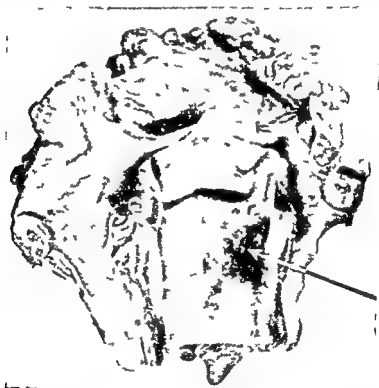


FIG 41 Laryngeal ulceration following use of cuffed tube (Belam and Zuck. *Anaesthesia*)

laryngeal inflammation and ulceration have followed intubation with a cuffed tube a pneumatic tourniquet effect having apparently occurred ^{1*}. It would seem that the risk is greatest when induced hypotension is being used. It should also be remembered that the pressure in the cuff rises in accordance with the difference between the temperatures of the operating theatre and of the patient. For example a rise in temperature from 15° C to 37° C will cause a

10 per cent rise in cuff pressure. Cases have also been known of a thin walled tracheal tube being compressed by the pressure of the cuff surrounding it, thus leading to respiratory obstruction

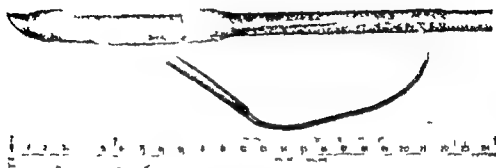


FIG 42 Low pressure cuff fitted to flexo-metallic endotracheal tube
(Bowen *Anæsthesia*)

The large cuff illustrated is designed to avoid these dangers and is made of latex fitted over a flexo-metallic tube the soft tip of which is reinforced with metal¹³. This cuff can be inflated by mouth to a very low pressure and an indicating balloon is not fitted to the inflation tube as even if a puncture exists leakage is slight. These tubes may require a lubricated introducer to facilitate intubation.

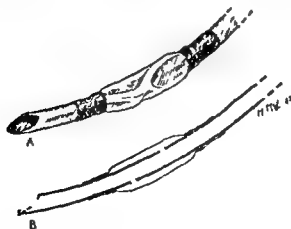


FIG 43 Self inflating cuff
A Deflated B Section showing cuff inflated (Mushin)

A self inflating cuff was designed for use with the Oxford vaporizer. This has no inflating tube, but several holes are cut in the endotracheal tube covered by it¹⁴. When the bellows of the vaporizer are compressed, the cuff inflates and makes a gas tight seal. On expiration the cuff deflates so that although controlled respiration can be maintained no protection is afforded against the aspiration of fluids.

Cardiovascular and Respiratory Disturbances during Intubation and Extubation

The passage of tracheal tubes under light general anæsthesia only can give rise to reflex disturbances of the respiratory and circulatory mechanisms and even death¹⁵. (This is not surprising since each year many instantaneous deaths occur in persons who have had food lodged on the glottis. These fatalities are not due to asphyxia as was once supposed but are reflex in origin¹⁶). Investigations have shown that electrocardiographic changes are very common at the moment of tracheal intubation¹⁷. For example in one series 68 per cent of all patients showed ECG disturbances during intubation but if intravenous procaine was given previously this figure was reduced to 24 per cent¹⁸. Cardiac arrhythmias are particularly likely to follow intubation under light cyclopropane and the barbiturates¹⁹ and are apparently caused by irritation of vagal fibres in the larynx and trachea although some workers believe that increased CO and decreased O₂ tension may be significant factors²⁰. Intubation carried out with the patient under the influence of suxamethonium appears to cause less cardiovascular disturbances than when other relaxants are used¹ (See also Chapter 7). Preliminary cocainization of the glottis carried out at least two minutes before intubation undoubtedly diminishes laryngeal reflexes and can be conveniently performed with one of the single handed sprays now available² (Fig 44). The risk of an absent reflex after



FIG 44 Single-handed laryngeal spray with small unbreakable container (Swerdlow *Lancet*)

extubation must, however be borne in mind if there is any possibility of fluid aspiration²³

After the removal of a tracheal tube, the patient must be kept under observation for a few minutes as cardiac arrhythmias are common²⁴ and actual arrest has been reported²⁵ Occasionally, owing to muscle spasm it may prove difficult or even impossible to remove a cuffed tube although complete deflation has taken place In this event a short acting relaxant should be administered²⁶ It sometimes happens, especially in children, that acute laryngeal obstruction occurs some hours after an endotracheal anaesthesia This is probably due to oedema of the sub glottic space, the mucosa of which is very sensitive in children²⁷ The condition may yield to palliative measures such as postural drainage and the inhalation of steam but in severe cases tracheotomy may be necessary and this is to be preferred to re intubation²⁸

Sterilization and Lubrication of Tubes

Tracheal tubes tend to lose their elasticity after frequent boiling or autoclaving and some other means of sterilization is advisable A usual and satisfactory practice in hospital is to clean the tubes internally with a brush of appropriate size and after washing to store them in a large enamelled bowl of 1 in 2000 biniodide of mercury Immediately before use they are rinsed in cold water and their distal 3 inches lubricated with a non oily preparation such as K Y jelly Oily lubricants such as liquid paraffin should be avoided as they cause rapid deterioration of rubber, but a castor oil emulsion seems to be satisfactory It is said that scrubbing the inside and outside of tracheal tubes for one minute each with 'phisohex' (a preparation containing hexylchlorophene) will sterilize them completely without causing mucosal irritation²⁹ Alternatively, the soaking of tracheal tubes for five minutes in 1 in 1000 zephiran solution is said to kill most organisms which may contaminate anaesthetic apparatus If a long endotracheal administration maintained at a light level of narcosis is contemplated, as during a craniotomy, an analgesic lubricant is helpful, a satisfactory one being xylocaine gel (2 per cent lignocaine in a water soluble base) A similar lubricant is xylodase, a 5 per cent lignocaine water soluble paste containing hyaluronidase to facilitate penetration of the mucosa by the local analgesic It is however a mistake to use long acting analgesics e.g. cinchocaine for comparatively short cases as the laryngeal reflex may be abolished for several hours

References

- 1 VISALIVS A 1542, *De Humanis Corporis Fabrica* 653
- 2 SNOW J 1858 *On Chloroform and Other Anæsthetics* 117 London
- 3 TRINDLENEBURG F., 1871 *Arch klin. Chir* 12 121
- 4 MACLEWEN W 1860 *Brit med J.*, 2 122.
- 5 ELSEBERG 1910 *N Y med Rec.*, March.
- 6 ROWBOTHAM E. S., and MAGILL, I W 1921 *Proc R Soc Med (An. Sec)*
- 7 WADDY F I 1954 *Brit med J.*, July 24 239
- 8 BLAIR GOULD R. 1954 *Brit med J* Sept. 11 645
- 9 BALLENTI E, R. I W., and JACKSON I 1954 *Anæsthesia* Jan. 4
- 10 BOUYNE, J G., 1947 *Brit med J* Oct 25 654
- 11 LAYCOCK J D., 1954 *Brit med J* Jan. 16 151
- 12 { STOUT R. J., and THOMAS C., 1954 *Brit J Anæsth.*, Jan., 35
- 12 { BELAM, O M and ZUCK, D., 1953 *Anæsthesia* April 96
- 13 { DARK, J., and JEWSEBURY P., 1955 *Lancet* Feb 26 430
- 13 BOWEN R. A., 1954 *Anæsthesia* Jan 40
- 14 MUSLIN W W and BAKER R. *Principles of Thoracic Anæsthesia* 137
- 15 { REID L C *et al.*, 1952 *Arch Surg* April 409
- 15 { HARRISON G., 1949 *Anæsthesia* Oct., 181
- 16 SIMPSON K., 1949 *Lancet* April 2 558
- 17 REID L. C., and BRACE D E., 19-0 *Surg Gynec Obstet* 70 157
- 18 BURSTEIN C L *et al* 1950 *Anesthesiology* May 299
- 19 BURSTEIN C and ROVENSTINE, E. A 1938 *J Pharmacol & Exp Thor.*, May 42.
- 20 DENSON J S., and JOSEPH, S I., 1954 *Anesthesiology* Nov., 650
- 21 JOHNSTONE, M 1955 *Anæsthesia* April 122
- 22 SWERDLOW M., 1955 *Lancet* May 14 1004
- 23 NEGUS V E. 1933 *Brit J Anæsth.*, July 177
- 24 CONVERSE, J G *et al* 1952, *Anesthesiology* March 163
- 25 { SCHUMACKER H B and HAMPTON L. J 1951 *J thorac Surg* Jan 48
- 25 { RAFFAN A W 1954 *Anæsthesia* April 116
- 26 CULLINGFORD D W J., 1954 *Brit J Anæsth* May 187
- 27 MCGUCKIN P 1954 *Brit med J* June 12 1380
- 28 PRENDIVILLE, J B and MIDDLETON H G 1954 *Brit med J.*, May 13 1126
- 29 McDONALD W L. J *et al* 1955 *Anesthesiology* 16 206
- 30 TATE N 1955 *Lancet* Nov 5 980
- 31 CZERMAY J N 1861 *New Sydenham Soc Selected Monographs* 17
- 32 BURNS T H E 1956 *Brit med J* Feb 25 440

CHAPTER 5

THE EXPLOSION RISK IN ANÆSTHESIA

IN the last few years the use by surgeons of electrical and other apparatus producing heat and sparks has greatly increased, and it behoves the anæsthetist to bear in mind the possible risk of fire and explosion. If such an accident should occur, it is quite possible that he will become involved in medico legal proceedings.

Incidence

One of the first reports of an anæsthetic explosion in Great Britain was in 1892³⁰

Twenty five years ago it was estimated that at least 100 cases of burns of the eyebrows, lips, pharynx, etc., occurred in Great Britain every year from ether explosions¹ while a recent calculation has shown that the risk of an explosion is about 1 in 485 870³⁷

In an American series of statistics compiled from the records of eighty seven anæsthetists, the explosion rates for ether, ethylene and cyclopropane were all in the neighbourhood of 2 to 4 per 100 000 anæsthesias, and the explosion mortality was 1 in 1 150,000 cases.

It will be convenient to discuss the matter under (a) the explosive mixture, (b) the source of ignition, and (c) means of separating (a) from (b).

The Explosive Mixture

The anæsthetic agents capable of igniting or exploding in suitable conditions are ethylene, acetylene, cyclopropane, ethyl chloride, and the anæsthetic ethers with the partial exception of trifluoroethyl vinyl ether. The mixture of oxygen, nitrous oxide or both with any of these agents will usually increase the violence of the explosion and in certain cases a catastrophic detonation can occur³. It should be particularly noted that nitrous oxide and ether without any air or oxygen are explosive, the necessary oxygen being derived from the breaking down of nitrous oxide. The liberation of the 'energy of dissociation' of nitrous oxide into nitrogen and oxygen adds to the violence of the explosion. Since ether vapour will ignite at 190° C⁴ and liquid ether contaminated with peroxides at 100° C, it will be realized that excessive heat is by no means necessary. Thus is cautery at a temperature of 300° to 350° C

below visible red heat may ignite an oxygen-ether vapour⁵ The limits of inflammability and explosibility of ether are from 3 per cent. to 100 per cent in oxygen⁶ and 3 per cent. to 34 per cent. in air The higher percentages of ether can ignite by means of a slowly travelling cool flame which is a particularly insidious type of ignition since it cannot be seen in daylight This 'cool flame' is initiated by hot wires but not by sparks⁷ Since the vapour density of ether is more than double that of air, any escaping vapour tends to run downwards in a concentrated stream so that ignition at floor level at some remote point is not impossible⁸ Cyclopropane has acquired an evil reputation owing to the very low energy of a spark required to ignite it For example a 4 per cent ether concentration requires a spark energy about 45 times greater than that which will ignite cyclopropane⁹

It is possible for explosions and fires to take place apart from the anæsthetic agent employed For example fatal accidents have occurred from use of a diathermic needle on skin which had just previously been sterilized with ether or spirit.

The detection of an inflammable or explosive gas mixture can be effected by an instrument known in America as a Vapotester¹ This consists essentially of a Wheatstone bridge with a balanced electrical circuit (Fig 45) The circuit includes two incandescent

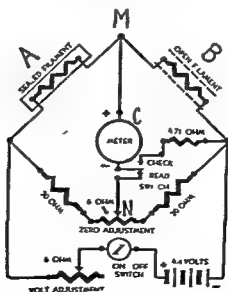


FIG 45 Diagram to show principle of "Vapotester" to ascertain if a gas mixture is inflammable (J W Uhl *et al* *Anesthesiology*)

platinum wires one of which (A) is enclosed while the other one (B) is situated in a test chamber through which a sample of the gas can be drawn. If this is combustible the temperature of the wire (B) rises and its electrical resistance is increased, thus unbalancing the circuit and showing a current on the ammeter (C) ¹⁰ (See Fig 45)

A simpler apparatus for determining if a sample of a gas mixture is inflammable or explosive consists of a 2 in length of 1 in steel rod drilled through and tapped at one end to take a sparking plug and turned at the other end to hold a toy rubber balloon. This fitting is fixed to a stand by a side tube through which the balloon can be filled with a sample of the gas mixture. A 6 volt battery, car induction coil, flex and switch complete the outfit ¹¹. This will convince the most sceptical surgeon of the inadvisability of using the diathermy on the air passages of a patient anesthetized with cyclopropane.

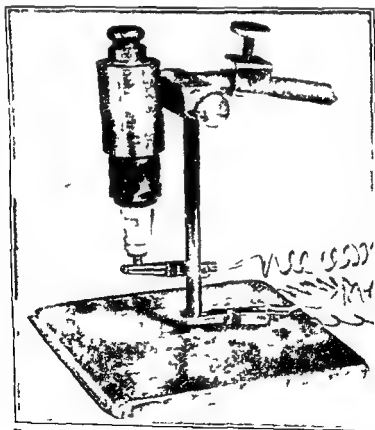


FIG 46 Morton's apparatus for testing explosive properties of anesthetic gases (*Brit med J*)

The non flammable inhalation anæsthetics are nitrous oxide, xenon⁴⁰ trichlorethylene chloroform and halothane⁴¹

Doubts have recently been cast on the safety of trichlorethylene and it may be wise to enlarge on the facts. Mixed with air the drug is completely safe from ignition risks. If mixed with air enriched with oxygen or with pure oxygen at a temperature exceeding 78° F (25.5° C) a concentration of over 10 per cent trichlorethylene has an ignition temperature of 786° F (419° C). It is extremely unlikely that these conditions would occur in anæsthetic practice³⁸

Lastly we have to consider inflammable gases generated within the body. It is well known that flatus in the lower bowel can burn or explode and several such accidents have been reported when the diathermic cautery was used in the absence of inhalational anæsthesia⁴². One means of avoiding this accident when the diathermy is used through a sigmoidoscope is to insufflate carbon dioxide down the barrel of the instrument through a fine tube carried to its distal end. It has also been established that in certain patients (particularly those with pyloric obstruction) the stomach gases contain enough methane and hydrogen to be combustible⁴³. Furthermore if diathermy is used incorrectly and arcing is permitted inflammable gases due to the decomposition of tissue are liberated and may collect in a confined space such as the mouth. Explosions have almost certainly been due to this cause also¹⁴

The Source of Ignition

The probable sources of ignition in an operating theatre are —

- | | |
|----------------------------|---------------------------|
| (a) Static electricity | (c) Other sources of heat |
| (b) Non static electricity | (d) Spontaneous ignition |

Static electricity In general it can be stated that the drier the atmosphere the more likely are static sparks to occur. It is a wise precaution to install hygrometers in anæsthetic rooms and operating theatres so that a check can be kept on the atmospheric conditions. Hygrometers depending on the length of a human hair are suitable for this purpose as the relative humidity can be read off from a dial. They must however be kept in one position and recalibrated fairly frequently. The wet and dry bulb thermometer type of hygrometer is more accurate but requires tables to convert the readings into humidity percentages. It was originally held that there was no risk of static sparks if the relative humidity exceeded 54 per cent⁴⁴ but more recently the higher limit of 60 per cent has been accepted⁴⁵. It would seem clear that an obvious way to avoid static charges

would be to increase the relative humidity and this is often done unintentionally by steam escaping from the sterilizing plant. Unfortunately however a high humidity leads to stuffy and unpleasant working conditions and in practice dehumidification is more likely to be attempted than the reverse¹

A slight ionization of the theatre atmosphere with β rays allows static charges to leak away rapidly as can be demonstrated by approaching a charged object with a small quantity of radio active material. It has been suggested that a slight ionization of the intake of air-conditioning systems might be a practical proposition.

The voltage of static charges is important as sparks are unlikely unless the difference in potential exceeds 350 volts¹⁷ and even then they are less than $\frac{1}{1000}$ in long and incapable of igniting an inflammable vapour. Voltages of 5000 to 15,000 however, have been recorded in operating theatres and the resulting sparks are extremely dangerous¹⁸. In general static sparks are of very short duration e.g. one millionth of a second and the discharge is oscillatory in character¹⁹. A portable instrument known as a statigun can be used to record the potential gradient in volts per foot and if held one foot from a charged object will record the voltage of the charge.²⁰ Another instrument known as a 'statometer' or 'staticater' has



FIG. 47 The Baldwin Dunlop statigun

been developed in America to record dangerous static charges. One model of this apparatus emits a buzzing sound when a charged object is within a certain distance of it.¹⁰

The incidence of explosions from static sparks varies so much with climatic conditions that actual figures are of little value. Until fairly recently Great Britain was regarded as almost immune from the danger but unfortunately several accidents have shown that this is not the case¹¹ and recently it has even been suggested that most explosions in operating theatres are due to this cause.¹²

The factors leading to the generation of static charges in operating theatres are varied, some of the commonest being the passage of gases through rubber pipes, the motion of rebreathing bags, the drawing of blankets over rubber topped trolleys etc. As regards textiles, woollen blankets and silk or nylon underwear, pillow cases, aprons and gowns are the worst offenders and cotton linen or viscose rayon should be substituted wherever possible.

A great deal of attention has been paid to the dispersal of static charges before they assume dangerous dimensions. The original idea of an 'intercoupler device' connecting electrically all persons and objects to a common earth originated in America but has never found favour in this country.²⁰ One of the reasons for this is that if a patient connected directly to earth comes into accidental contact with a mains current (e.g. 240 volts A.C.) he is more likely to be seriously injured or killed than otherwise. The modern practice is to have all relevant objects connected to earth through a moderate resistance. In actual fact this means the use of suitable flooring for anæsthetic rooms and theatres and the substitution of anti static rubber for the ordinary variety.

Most modern operating theatres have floors of the 'terrazzo' type in which a matrix of marble chips is made solid with magnesium oxychloride and then ground smooth. Such floors have sufficient conductivity to prevent the accumulation of dangerous static charges if all the equipment is fitted with chains which trail for a few inches or with wheels on which are anti static tyres. Non conducting rubber flooring, linoleum, cork carpet and wood blocks on the other hand, have a high resistance and unless metal strips are sunk every few inches it is impossible for earthing devices to disperse charges effectively.⁸ A committee was recently set up in America to make recommendations for theatre floors in Governmental hospitals.² It recommended *inter alia* that floors should have a resistance of less than 500 000 ohms as measured between two electrodes 3 ft. apart. The resistance should also be more

than 25,000 ohms as measured between two electrodes placed 3 ft apart at any location on the floor and more than 3 ft from any earthed object. In this country the Ministry of Health regulations call for resistance dry not greater than 2 million ohms and when wet not less than 100 000 ohms. The mopping of high resistance floors with soapy water, the laying down of damp sheets or polishing them with anti static carbowaxes will increase their conductivity appreciably for some time.* This practice might well be considered for temporary operating theatres.

The important question of rubber must now be considered. Until fairly recently ordinary red or black rubber was used for many purposes in anæsthetic apparatus and theatre furniture. Rubber of this variety has two dangers. It can itself generate high static charges on movement, e.g. the emptying and filling of rebreathing bags, and it prevents charges from escaping by its insulating properties. The idea of producing conductive rubber is not a new one as a British patent for its manufacture with graphite was granted in 1882.²³ The matter was not pursued however until the danger of charges on aircraft forced the development of anti static tyres for landing wheels. Conductivity is achieved by adding finely divided carbon to the rubber mix.²⁴ This addition is known as 'carbon black' the purest variety being acetylene black containing 99 per cent carbon. This substance is usually made by the continuous decomposition of acetylene at a temperature of over 1500° C ($C_2H_2 \rightarrow 2C + H_2 + \text{heat}$). The light fibrous material thus formed is compressed into one of two bulk densities weighing 25 lb per cu ft (50 per cent) or 12.5 lb (100 per cent compressed black). A typical formula for anti static rubber mix is:⁵ rubber 60.5, sulphur 2.25, zinc oxide 4.5, stearic acid 2.0, paraffin wax 2.0, anti oxidant 0.5, mercaptobenzothiazole 0.75, acetylene black 28.0. The usual resistance of anti static rubber lies within the range of 10^6 – 10^8 ohms (1–100 megohms). This is sufficient to act as an insulator for mains circuits of 240 volts but disperses static charges.²⁵ The term "conductive rubber" is properly applied to material with a resistance of less than 105 ohms. During the past few years anti static rubber has been substituted for the ordinary variety in anæsthetic equipment and theatre furniture such as mattresses, trolley wheel tyres, goloshes, etc.* Thus static charges tend to leak away via the floor and it has been found that anti static tyres are much more efficient than the trailing chains which they have largely replaced and which

* It is worth noting that if the theatre staff are not provided with anti static rubber shoes, leather soles are considerably safer than standard rubber soles.²⁶

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The use of endoscopes fitted with electric lamps calls for care. In order to provide maximum illumination such lamps are often grossly overloaded with consequent rise of their outside temperature. It is recommended that such lamps should always be lit from batteries whose maximum voltage does not exceed the rated voltage of the lamps by more than 25 per cent.⁷ This is very much safer than obtaining the current from A C mains with a transformer. It is most important that all electrical wiring of endoscopes should be regularly inspected for short circuits and poor connections.

Electrical apparatus some way from the site of operation must be spark proof as in certain circumstances inflammable vapours can travel for a considerable distance. The armatures of electric motors driving suction pumps or bone saws are not always above suspicion, nor are the spark gap interrupters of some diathermy apparatus. Although modern X ray apparatus is said to be spark-proof it is unwise to use an inflammable vapour while the plant is working. Many explosions have occurred with obsolete plant.⁸

In modern operating theatres the electrical switchgear should be laid out with a view to rendering sparks impossible. Not only should the switches themselves be spark proof but they should be fitted with interlocking adapters so that the flexible leads cannot be removed with the current on. The switches should be at least 3 ft from floor level to avoid contact with heavy layers of inflammable vapour.

It is not generally recognized that an incredibly small spark energy can ignite gas mixtures. For example 10 ergs can ignite cyclopropane or ether air mixtures and if converted into heat this amount of energy would only suffice to raise the temperature of 1 ml of water by one five millionth of a degree Centigrade.¹²⁹

Other Sources of Heat When operations are to be performed in private houses it is necessary to make sure that the heating of the rooms is not effected by means of open fires, gas stoves or electric radiators with exposed elements. Some old fashioned sterilizers are still operated by naked flames from spirit stoves or gas rings. In dental practice the operator sometimes dries a cavity by blowing hot air into it and this has resulted in an explosion of ether vapour in the mouth. Small sparks are occasionally visible when dental forceps slip on the enamel of teeth and it has been proved that these can ignite diethyl ether, di vinyl ether and cyclopropane.³⁰

Spontaneous Ignition If oil or grease comes into contact with highly compressed oxygen a spontaneous fire may start which may

gradually acquired an insulating coating of dust, grease etc. It is worth noting that the painting of ordinary rubber with a high grade aluminium paint gives a temporary surface of fairly high conductivity. Another way of achieving greater safety in anæsthetic apparatus fitted with ordinary rubber components is to use the CO₂ absorption technique. In a short time the saturation of the respired gases with water vapour will cover the inside of the corrugated tubing and rebreathing bag with a conductive layer of moisture which should do much to avoid dangerous local charges of static electricity from being built up. Even this is not a sure safeguard as one of the authors has seen an explosion which proved fatal to the patient due to the generation of static electricity in a circuit which had been closed for over an hour, cyclopropane being the anæsthetic in use. If an apparatus has been unused for some time it is wise to rinse out the tubing and rebreathing bag with soapy water before the induction of anæsthesia. If it becomes necessary to make or unmake metallic connections during narcosis a change over to a non inflammable agent should be made *some time before* the event. Unfortunately anti static rubber is considerably less flexible than the ordinary variety and this is particularly disadvantageous in the case of rebreathing bags in which dangerous pressures can readily be attained.²⁷ The latest products of manufacturers however show some improvement in this respect. In this country anti static rubber is black with distinctive red or yellow markings.

Non static electricity. Certain operative procedures such as use of the diathermic cautery unavoidably provide a source of ignition, and in such cases it is the anæsthetist's duty to avoid any possibility of inflammable vapours coming into contact with it. At one time it was thought to be an adequate precaution if inflammable vapours were confined to a closed circuit provided that the surgeon was using the diathermy at some point remote from the face piece and not in contact with the patient's respiratory passages. This is definitely not the case as if an imperfect earth is provided by the indifferent electrode e.g. by insufficient wetting it is quite possible for a current to pass through an alternative path such as an earth via the face piece and anæsthetic apparatus. Many surgeons and anæsthetists do not realize that diathermy currents are often at a voltage of 4000 and are at radio-frequency consequently they do not behave like comparatively low voltage D.C. or A.C. currents.²⁸ There is no doubt that surgical diathermy is a potentially dangerous proceeding which while it may be extremely valuable in certain cases, is often used unnecessarily and with little justification.

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Spontaneous Ignition If oil or grease comes into contact with highly compressed oxygen a spontaneous fire may start which may

prove impossible to extinguish. For this reason the greatest care must be taken to ensure that the screw threads and washers of oxygen cylinders are dry. Leather washers should never be used as it is difficult to free them entirely from grease. Fibre washers are safe, but so hard that gas tight joints are sometimes difficult to secure. The most satisfactory washer to date is constructed of lead in a brass casing which prevents spreading of the soft metal.

Reducing valves are a potential source of danger, one of the writers having had a narrow escape when a valve blew to pieces. The following precautions are wise when dealing with these fittings.

(1) they should be so positioned that they point at the floor or a blank wall,

(2) they should be fitted with some type of safety blow off,

(3) any tap or fine adjustment valve should not be rigidly connected to the outlet but at the distal end of a length of rubber pressure tubing,

(4) cylinders should be turned on gently with the outlet taps open (if fitted),

(5) separate reducing valves should be kept for different gases, e.g. a valve used on a nitrous oxide cylinder should never subsequently be used for oxygen.

Separation of the Explosive Mixture from the Source of Ignition

As a general principle it is sound practice to avoid explosive anæsthetic mixtures if there is any source of ignition present. This will exclude the use of ethyl chloride, the ethers and the hydrocarbon gases. It has been estimated that a slight leak of an inflammable mixture in a closed system will not result in an explosive risk at a distance exceeding 1 ft. from the leak.²¹ If the leak is extremely small, e.g. round the joint between the corrugated breathing tube and its metal connection, the gas may burn without explosion. On the other hand, gas mixtures escaping from quite small holes less than 1 square millimetre in area may ignite and light back to a closed or semi-closed circuit causing a disastrous explosion with grave injury to the patient.²²

The modern practice of using a closed circuit with fairly high gas flows and an intentional leak can be used without increased risk if the excess gases are led off through a pipe terminating outside the theatre. A special tap and T piece have been designed for this purpose and these are equally suitable for a semi-closed circuit if the expiratory valve is kept shut and the distension of the rebreathing bag is controlled by means of the tap.²³

It need hardly be said that diathermy of lung tissue or indeed anywhere inside the pleural cavity is not permissible if an inflammable

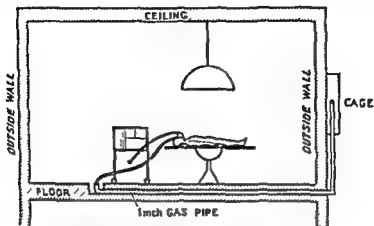


FIG 48 Diagram showing method of removing explosive gases from theatre (Bullough *Lancet*)



FIG 49 T junction in use with semi-closed circuits (Bullough *Lancet*)

anæsthetic is being used. The possibility of a bronchial fistula must also be borne in mind. If a closed system is not available ether vapour can be adsorbed by passing the expired gases through activated charcoal. In an emergency the container of a service

respirator is efficient for a short time, but for prolonged periods a larger container such as a Waters canister is necessary³¹

Explosions have occurred when the anæsthetist has imagined that he was administering a non inflammable vapour e.g. nitrous oxide oxygen-chloroform. Such accidents are invariably due to faulty apparatus, as it is possible in some cases for an explosive mixture containing ether to be delivered although the indicator is set at 'chloroform'.³⁵ This may be due to positive pressure in the ether bottle from a hot water jacket or to a partial vacuum occurring from the gases passing over it.³⁶ If there is any doubt of the correct functioning of such apparatus the ether bottle should be removed entirely when an ignition risk is present.

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Finally it is necessary to keep a sense of proportion. Explosions are spectacular and give rise to much publicity but actually they are rare and many other factors affecting the safety of the patient may have much more importance in any given case.

References

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- 6 THORNTON W M 1939 *Proc Instn Elect Engrs* Feb
- 7 SWANN H W 1938 *Brit med J* July 30 234
- 8 RAYNER, E. H 1928 *Proc Instn Elect Engrs* Feb
- 9 JARMAN R 1948 *Proc R Soc Med (An Sec)* March 5
- 10 UHL, J W *et al* 1949 *Anesthesiology* July 479
- 11 MORTON H J V 1951 *Brit med J* Feb 10 298
- 12 { LIEBERMAN W 1944 *Rev Gastroent* 2 259
- 12 { MARCHAND J H 1946 *Arch Mal Appar Dig* 35 240
- 12 { MOUTIER, F *Arch Mal Appar Dig* 35 242
- 13 EAST T 1934 *Lancet* 2 252
- 14 { HAMBLETON H S *et al* 1935 *J Amer med Ass* 105 645
- 14 { GALLEY A H., 1954 *Brit J Anæsth* May 189
- 15 ROVENSTINE, E. A 1936 *Amer J Surg* Dec 460
- 16 BULGIN D *et al* 1949 *Lancet* 1 789
- 17 SILSBEE, F B., 1942 *U.S. Standards Bureau Circ* C 438 36
- 18 THOMAS C J 1950 *Amer Soc Anæsth Newsletter* No 8
- 19 LOW W A 1950 *Proc R Soc Med (An Sec)* Dec 1
- 20 BRACKEN A 1952 *Med Illus* Dec 643
- 21 { IRONSIDE, R., 1935 *Proc R Soc Med (An Sec)* March 1
- 21 { CHIVERS E. H 1943 *Lancet* April 24 527
- 22 { Rep Cte on Explosions in Hospital Operating Suites 1950 Jan 1
- 22 { (Washington, D.C.)
- 22 { Bureau of Mines Rep of Investigation 4833 1952, Jan

- 23 British Patent 6767
- 24 Annotation 1947 *Lancet* April 5 469
- 25 MANTELL, C. L. 1946 *Industrial Carbon* 2nd ed New York
- 26 WOODLAND P C and ZIEGLER E. E *Modern Plastics* 28 95
- 27 ROBSON J G 1954 *Brit med J* May 1 1040
- 28 GREENE, II A 1952 *Anesthesiology* March 203
- 29 ROBERTS J E. and HEWER A J II 1953 *Anæsthesia* April 79
- 30 { BOURNE J ■ 1954 *Brit med J* April 17 937
- 31 { BOURNE, J G and MORTON H J V 1955 *Lancet* Jan 1
- 32 { HORTON J W 1941 *Anesthesiology* March 122
- 33 { HEWER, C. L. 1930 *Proc R Soc Med (An Sec)* April
- 34 { GREENE, II A 1941 *Amer J Roentgenol* 45 737
- 35 BULLOUGH J 1954 *Lancet* April 17 798
- 36 EPSTEIN H G 1944 *Lancet* Jan 22, 114
- 37 FEATHERSTONE, H W 1931 *Proc R Soc Med (An Sec)* Nov 5
- 38 PRIMROSE, W B 1939 *Brit med J* Feb 4 215
- 39 Min of Health Rep 1954 Part II 168 H.M.S.O.
- 40 Report of Working Party on Anæsthetic Explosions 1956 H.M.S.O.
- 41 KRANTZ, J C. et al 1953 *J Pharmacol* Aug 488
- 42 BRACKEN A et al 1956 *Anæsthesia* Jan 40

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- 14 { HAMBLETON B. III *et al* 1935 *J Amer med Ass* 105 645
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Another method of using the continuous drip to produce anaesthesia and relaxation has been described⁶ and utilizes the short-acting relaxant suxamethonium chloride and the analgesic lignocaine. Induction is by thiopentone 500 mg and suxamethonium chloride 50 mg given intravenously from separate syringes. After hyper ventilation with oxygen a Magill cuffed tube previously lubricated with 5 per cent lignocaine ointment is inserted into the trachea. Ventilation is by nitrous oxide and oxygen in the proportion of 5 : 2. A continuous drip of suxamethonium chloride 0.1 per cent (500 mg in 500 ml of 5 per cent dextrose) and another of 0.5 per

CHAPTER 6

INTRAVENOUS ANÆSTHESIA

Intra-arterial Injection—Extravascular Injection—Intravenous Codein—Thiopentone and Gallamine Drip—Lignocaine and Suxamethonium Drip—Barbiturate Narcosis and Porphyria—Thiamylal—Sodium Seconal—Buthalitone Sodium—Hydroxydione (Viadril)—Thialbarbitone—Pharmacology of Thiopentone—Nisentil—Intravenous Bromethol—Sodium Succinate—Procaine—New Apparatus

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Anæsthetists are becoming more and more aware of the great dangers which may follow the intra-arterial injection of thiopentone and some workers of experience teach that veins near the bend of the elbow should always be avoided. If in spite of every care some of the drug is accidentally injected into an artery the needle should be left in the lumen of the vessel and used for intra arterial injection of procaine, papaverine or tolazoline (priscol). 5 ml of a 1 per cent solution. One of these drugs can also be injected into the subclavian artery following the injection to produce a block of the brachial plexus or the stellate ganglion.

Extravascular injection of thiopentone is always less serious if a 2.5 per cent solution is routinely substituted for the customary 5 per cent solution. A sterile gallipot and some normal saline or distilled water are the only additions to the anæsthetist's armamentarium for routine use. A mixture of procaine and hyaluronidase into the area will reduce post operative pain and discomfort by diluting the irritant alkaline thiopentone solution.

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more potent than an equal dose of thiopentone. On the other hand a blind study¹¹ involving 1098 thiopentone and 1245 thiamylal anaesthetics in humans, under routine conditions, subjected to statistical analysis of the effects of the drugs showed that with regard to potency, cardiotoxicity, respiratory depression, incidence of laryngospasm and recovery time no statistically significant differences existed. Dundee and Riding¹² reported their observations on 1750 patients who received the drug and concluded that it is a safe intravenous anesthetic very similar to thiopentone but of slightly greater potency and with less tendency to cause cumulation. A further report on this drug is that of Lund,¹³ who used it in no less than 6000 consecutive cases. His conclusion coincides with that of the other authors and he regards it as a safe and effective intravenous agent for anaesthesia. This opinion has been corroborated by Phillips¹⁴ who states that it is more rapidly detoxicated than thiopentone.

Sodium seconal has been used by Bryce Smith and Hingson¹⁵ and these writers conclude that it is an effective sedative to use in all forms of regional analgesia provided that painful impulses are completely blocked. Because of its longer and less intense action it is useful for induction of anaesthesia of patients in bed. When carefully given it can be used in subjects with heart, liver and kidney disease. These authors think well of it as an agent in endotracheal intubation when accompanied by adequate topical laryngeal analgesia. Its somewhat prolonged action makes it unsuitable for ambulatory patients, while its analgesic effects are almost nil. Used alone in labour it is unsatisfactory, but when combined with scopolamine, pethidine etc. it is a useful drug.

Buthalitone sodium (transithal, baytenal, ulbreval, thalbutone) is a short acting barbiturate having the chemical constitution sodium 5 allyl 5-iso butylthiobarbiturate. It was first synthesized some years ago by Miller and his colleagues,⁴ but interest was recently revived in it by Weese and Koss.²⁴ It is designed for intravenous injection as a 5 per cent or 10 per cent solution and remains stable for 36 hours in solution. It contains 6 per cent sodium carbonate to enhance its stability in solution. The first report of its use in Britain was that by Nobes in 1955.²⁵ Its sedative effects do not last as long as those of thiopentone so that it is especially recommended together with gas and oxygen for operations which are not expected to last for longer than 30 minutes. Soon after its effects have worn off the patient is clear headed, free from drowsiness and is able to look after himself safely. A total dose greater than 1 g. is seldom necessary,

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cent lignocaine isotonic with saline is prepared and run into one needle in a vein by means of a Y piece, enabling the rates to be controlled independently. To begin with, 60 drops a minute for 3 minutes and then 40 drops a minute are run in which is about 12.5 mg a minute or 750 mg an hour of lignocaine. After 40 minutes the dosage is reduced to 20 drops a minute and afterwards if necessary to 10 drops a minute. Suxamethonium chloride is run as a fast drip until the patient ceases to breathe when controlled respiration is established. After the initial exploration of the abdomen the rate is reduced to allow relaxation in the presence of normal respiration. The results reported were good and neither the blood pressure nor the pulse rate tended to alter. Post operative analgesia was a marked feature, but very occasionally a convulsion occurred due to overdosage. Although both drugs appear to be hydrolysed by pseudo cholinesterase no prolonged apnoea was seen while the lignocaine seemed to potentiate the relaxant properties of suxamethonium. Return of reflexes and consciousness was rapid. The authors warn against the combination of hexamethonium and lignocaine as the hypotension produced by the former is likely to be prolonged. Nausea and vomiting were reduced and it is suggested that intravenous lignocaine may have a useful place in the management of such cases as severe burns.

The problem of large scale sterilization of syringes has been solved in various ways. Burns⁸ described an improved syringe container. It is a glass tube for the sterilization of a syringe with its needle, and is procurable from Down Brothers, London.

Intravenous Barbiturates

Substitutes for thiopentone appear from time to time, one of the latest being thiamylal (thioquinalbarbitone sodium, thioseconal sodium, surital sodium). In this drug the allyl group replaces the ethyl group of the thiopentone molecule. It is chemically and physically very similar to thiopentone and bears the same relationship to quinalbarbitone (seconal) that thiopentone does to pentobarbitone (nembutal). It was described by Volwiler and Tabern in 1935. A comparison of the potencies of thiamylal and thiopentone has been reported by Mushin and his colleagues⁹ who found it to be slightly more potent than the older drug. Jolly and Lederer¹⁰ found thiamylal to be superior to thiopentone as it seemed to cause less respiratory depression and interference with the peripheral circulation. Laryngeal spasm and gagging were less noticeable and recovery more rapid than after thiopentone. They found the drug

more potent than an equal dose of thiopentone'. On the other hand a blind study¹¹ involving 1098 thiopentone and 1245 thiamylal anaesthetics in humans under routine conditions, subjected to statistical analysis of the effects of the drugs, showed that with regard to potency, cardiotoxicity respiratory depression, incidence of laryngospasm and recovery time no statistically significant differences existed. Dundee and Riding¹² reported their observations on 1750 patients who received the drug and concluded that it is a safe intravenous anaesthetic very similar to thiopentone but of slightly greater potency and with less tendency to cause cumulation. A further report on this drug is that of Lund,¹³ who used it in no less than 6000 consecutive cases. His conclusion coincides with that of the other authors and he regards it as a safe and effective intravenous agent for anaesthesia. This opinion has been corroborated by Phillips¹⁴ who states that it is more rapidly detoxicated than thiopentone.

Sodium seconal has been used by Bryce Smith and Hingson¹⁵ and these writers conclude that it is an effective sedative to use in all forms of regional analgesia provided that painful impulses are completely blocked. Because of its longer and less intense action it is useful for induction of anaesthesia of patients in bed. When carefully given it can be used in subjects with heart liver and kidney disease. These authors think well of it as an agent in endotracheal intubation when accompanied by adequate topical laryngeal analgesia. Its somewhat prolonged action makes it unsuitable for ambulatory patients while its analgesic effects are almost nil. Used alone in labour it is unsatisfactory, but when combined with scopolamine, pethidine etc. it is a useful drug.

Buthalitone sodium (transithal, baytenal, ulbreval, thalbutone) is a short acting barbiturate having the chemical constitution sodium 5 allyl 5 iso butylthiobarbiturate. It was first synthesized some years ago by Miller and his colleagues⁴ but interest was recently revived in it by Weese and Koss²⁴. It is designed for intravenous injection as a 5 per cent or 10 per cent solution and remains stable for 36 hours in solution. It contains 6 per cent sodium carbonate to enhance its stability in solution. The first report of its use in Britain was that by Nobes in 1955²⁵. Its sedative effects do not last as long as those of thiopentone so that it is especially recommended together with gas and oxygen for operations which are not expected to last for longer than 30 minutes. Soon after its effects have worn off the patient is clear headed free from drowsiness and is able to look after himself safely. A total dose greater than 1 g is seldom necessary.

cent lignocaine isotonic with saline is prepared and run into one needle in a vein by means of a Y piece, enabling the rates to be controlled independently. To begin with, 60 drops a minute for 3 minutes and then 40 drops a minute are run in, which is about 12.5 mg a minute or 750 mg an hour of lignocaine. After 40 minutes the dosage is reduced to 20 drops a minute and afterwards if necessary to 10 drops a minute. Suxamethonium chloride is run as a fast drip until the patient ceases to breathe, when controlled respiration is established. After the initial exploration of the abdomen the rate is reduced to allow relaxation in the presence of normal respiration. The results reported were good and neither the blood pressure nor the pulse rate tended to alter. Post-operative analgesia was a marked feature, but very occasionally a convulsion occurred due to overdosage. Although both drugs appear to be hydrolysed by pseudo-cholinesterase no prolonged apnoea was seen while the lignocaine seemed to potentiate the relaxant properties of suxamethonium. Return of reflexes and consciousness was rapid. The authors warn against the combination of hexamethonium and lignocaine as the hypotension produced by the former is likely to be prolonged. Nausea and vomiting were reduced and it is suggested that intravenous lignocaine may have a useful place in the management of such cases as severe burns.

The problem of large-scale sterilization of syringes has been solved in various ways. Burns⁸ described an improved syringe container. It is a glass tube for the sterilization of a syringe with its needle and is procurable from Down Brothers, London.

Intravenous Barbiturates

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oxygen and pethidine as the maintenance agents, a smooth anaesthesia results and pharyngeal and laryngeal reflexes are depressed and the incidence of laryngeal spasm lessened. Laryngoscopy is not actively resisted either by the larynx or by the muscles of the patient's body once sleep is induced. ■ after 500-700 mg has been infused. Its fate in the body is not known. When slowly injected 0.5 ■ causes drowsiness while 1 g usually produces deep unconsciousness which may persist for 1 to 2 hours. It is more of a hypnotic than an anaesthetic, and causes very little abdominal relaxation. It gives rise to similar electro-encephalographic patterns as such barbiturates as pentobarbitone, hexobarbitone and thiamylal.⁴⁰ Occasionally it results in a steep fall in the blood pressure but no cardiac arrhythmia has been observed.⁴¹ Hypotension is readily reversed by a small dose of a vasopressor drug. It produces less respiratory depression than thiopentone and gives rise to no parenchymatous changes. Post-operative vomiting is rare and a feeling of well being rather than a hangover is experienced in the hours immediately after anaesthesia.⁴² The low blood pressure may be due to inhibition of the vasoconstrictor centre, coupled with a peripheral vasodilatation.⁴³ The drug may also directly depress the myocardium. It often gives rise to ■ tachycardia.⁴⁴⁻⁵⁰

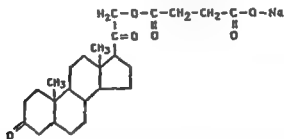
Side effects include twitching⁵¹ and venous thrombosis which is not uncommon. It may prove useful in laryngoscopy and in bronchoscopy in association with topical analgesia, but the slow onset of anaesthesia and the prolonged recovery period are definite disadvantages.

Thialbarbitone (kemithal) has been reported by Harrison¹⁶ as producing arrhythmia to a greater extent than thiopentone. She investigated ■ large number of cases and took careful ECG's in a series of them.

Many newer intravenous barbiturates are described but so far in this country none has yet displaced that old trusty thiopentone. Inactin (sod 5 ethyl 5 methyl propyl 2 thilbarbitone) is similar to thiopentone and was described by Tabern and Volwiler in 1935. Thionarcex (sod 5 ethyl 5 butyl ethyl 2 thiobarbitrate) also known as JL1014 is slightly more potent than thiopentone with a slightly more rapid recovery time. Thiogenal (neraval sodium) is sodium methyl thioethyl 2 pentyl thiobarbiturate another new short acting drug. It ■ said to have slightly greater parasympathomimetic properties and ■ more rapid in its action than thiopentone.⁵¹⁻⁵

while less than 0.5 g is likely to be insufficient in a healthy adult. For dental extractions a suitable dose is 0.6 g for a man and 0.4 g for a woman together with gas and oxygen. The drug closely resembles hexobarbitone in its effects but is more rapidly destroyed, probably in the liver. It is said to be useful in the casualty department and in the out-patient clinic, in the dental chair and in obstetrics for delivery of the head. Its solutions are readily miscible with gallamine and with suxamethonium halides, but in the latter case injection must immediately follow preparation of the mixture. Like thiopentone it is occasionally followed by side effects such as coughing, hiccup, apnoea, clonus and hypotension.⁴³

Hydroxydione (viadril) is a non-barbituric anæsthetic for intravenous use, which has the chemical composition 21-hydroxypregnane-3,20-dione sodium succinate. The anæsthetic potency of a series of steroid compounds was demonstrated by Selye in 1941⁴² but it was not until 1955 that Laubach, Pan and Rudel in the United States advocated the use of hydroxydione as an anæsthetic.⁴⁴



Hydroxydione

Its first clinical employment was described by Murphy and his colleagues of the University of California.⁴ Hydroxydione is a non-volatile but readily soluble crystalline solid. Its solution is incompatible with d-tubocurarine, suxamethonium and pethidine.⁴⁵ A 5 per cent solution in 5 per cent dextrose has a pH of 7 but even in this strength it tends to irritate the walls of veins. It has neither androgenic nor estrogenic properties and the LD₅₀ in mice is three times that of thiopentone. It is supplied in 0.5 g ampoules and is probably best given as a 0.5 per cent solution in dextrose into a major limb vein. The smaller veins should be avoided because of its tendency to cause thrombophlebitis. The drip-rate for induction of anaesthesia is about 150 per minute and 1 g lasts about two and a half hours.⁴⁶ After injection the full effects are not seen for 10 to 20 minutes. The drug is not suitable as the sole anaesthetic but with pethidine and scopolamine as the premedication and with gas and

body weight is a factor, though not a striking one, in determining the dosage required in healthy adult patients. Males are found to be more tolerant than females to the drug. Requirements of thiopentone are fairly constant in middle age but are increased in those less than 25 and decreased in those more than 46 years of age.

The cumulative action of thiopentone has been investigated⁸ in a careful clinical and laboratory study and it is emphasized that the drug is slowly de-toxicated in the body; its short duration of action after small doses being due mainly to its rapid diffusion to the non-nervous tissues. Gradual saturation of the body's fatty deposits takes place next until finally there is complete body equilibrium. Lowering the pH of the plasma prolongs the time taken to saturate the fatty tissues. This is well seen after hypoventilation. Evidence has been produced that there remains in the body for 24 to 30 hours after the injection of a small amount of thiopentone sufficient of the drug to affect the duration of a subsequent dose so that extreme care to avoid over dosage is necessary if thiopentone is administered within a short time of a previous injection.

Hæmodynamic studies during anaesthesia with thiopentone and gas and oxygen have been made.⁹ These studies were conducted in patients in whom the depth of anaesthesia was carefully controlled by EEG monitoring. It was found that cardiac output decreased with increasing depth of anaesthesia and that return to normal values was slow. As would be expected a decrease in blood pressure occurred during induction and was greater after rapid than after slow induction. A method of reversing the myocardial depression caused by thiopentone is said to be the intravenous injection of 5-10 ml of 10 per cent calcium chloride solution, given slowly. Thus while preventing a fall in blood pressure does not interfere with the duration or depth of anaesthesia.¹⁰ A decrease in arterial oxygen saturation was observed during induction with the patient breathing room air but if he breathed 100 per cent oxygen this did not occur.

Barbiturate Narcosis in Porphyria

Dundee and Riding⁷ in an original article point out the harmful effects of barbiturates especially thiopentone on porphyria and the advice is given that in patients suffering from any manifestation of this disease thiopentone is absolutely contra-indicated. They note that the disease may be congenital or familial with photo-sensitivity and pigmentation of bone and teeth. It may be confined to the skin with photo-sensitivity and recurrent bullous lesions due to

The Pharmacology of Thiopentone

Much work has been done on the pharmacology of thiopentone and the reader is referred to the excellent and comprehensive monograph by J W Dundee on Thiopentone and other Thiobarbiturates⁶⁷ Myers and his colleagues¹⁷ on experiments with dogs, ligated the portal vein to the hepatic artery in animals with an Eck fistula, so eliminating liver function. It was shown that the liver is by far the most important tissue in the detoxication of thiopentone in the dog. The effect of azotæmia on barbiturate anæsthesia has been investigated by Dundee and Roberts¹⁸. They remark that there is evidence to show that the kidneys possess some ability to detoxicate thiopentone in experimental animals with azotæmia. Longer narcosis is produced by a given dose of thiopentone than in animals with a normal blood urea. Hexobarbitone shows this change to a lesser degree. From clinical observations they state that uræmic patients require less thiopentone than do normal patients, and on the other hand normal patients who are caused to have a high blood urea level also require less thiopentone than the same patients when their blood urea is normal. The effect of thiopentone on the blood sugar is stated to be minimal provided the dosage is less than 1.5 g.¹⁹

Thiopentone as a factor in the production of liver dysfunction has been investigated.⁹ There is strong evidence that the liver is the main site of detoxication of thiopentone both in animals and man. 10 to 15 per cent of the drug in the body being metabolized each hour. In the light of this investigation it would appear that liver dysfunction occurs in an appreciable number of patients when doses of thiopentone exceeding 750 mg. are given and the incidence of liver damage is related to the dose of thiopentone. It is recommended that minimal doses should always be employed in known cases of liver dysfunction and in cases with thyrotoxicosis, acute intestinal obstruction, pulmonary tuberculosis or when a fall in blood pressure during the operation is expected. In a later paper²¹ it is shown that in the dog in a condition resembling uræmia in man narcosis with thiopentone and other medium acting barbiturates is prolonged.

Dundee² has also investigated the relationship of the patient's weight, sex and age to thiopentone dosage. He starts out by enumerating those pathological factors which influence the dosage of thiopentone, which include the unusual sensitivity to the drug shown by patients with liver damage, severe anæmia, malaria, shock and uræmia, and unusual resistance in those receiving large daily doses of sedatives or analgesic drugs. His investigations showed that

morphine giddiness being that most frequently seen. Its respiratory depressant properties are counteracted by nalorphine.

Siker, Foldes and their colleagues²⁵ used nisentil to supplement thiopentone nitrous oxide oxygen anaesthesia in 750 cases, using 600 patients given pethidine as a control group for comparison. When the nisentil was used in reasonably small amounts it enabled calm conditions to be maintained with smaller doses of thiopentone. More patients responded to stimulation at the end of the operation and depth of anaesthesia was more controllable, without undue depression of respiration, than when pethidine was used. In larger doses respiratory depression became an undesirable factor. The initial dose was usually between $7\frac{1}{2}$ and 15 mg, the safety interval between doses being 8 to 20 minutes.

Pethidine and gallamine alone have been used to facilitate the reduction of a fractured mandible. The difficulties and dangers and disadvantages of blind nasal intubation are well known and an alternative technique has been described.⁶ After premedication the anaesthetist explains to the patient that he will feel sleepy but will not lose consciousness and his co-operation is expected. With a suitable signalling guide to indicate his respiratory difficulty, pethidine is injected intravenously in fractional dosage using an in-dwelling needle. Usually 50 to 100 mg are required. Gallamine is at first given in 20 mg doses until the patient can no longer raise his head from the pillow. Additional doses of the two drugs are given as required, neostigmine and oxygen being readily available.

Bromethol is not in fashion these days, but Dwyer and his colleagues have reported their results following intravenous injection.²⁷ Bromethol is well known to produce certain effects useful to the anaesthetist. It causes sleep without restlessness, reduces the likelihood of vomiting, depresses upper respiratory tract reflexes and dilates the bronchi. On the other hand it causes hypotension, respiratory depression, intestinal stasis and hyperglycaemia. It is contra-indicated in patients with liver and kidney dysfunction and severe cardiac and pulmonary disorder etc. For intravenous work a 1 per cent solution in 5 per cent dextrose is employed and is tested with Congo Red. The drip is allowed to run rapidly until the patient is asleep and the rate is then suitably adjusted. 75 to 100 ml is the average amount required to produce sleep but up to 300 ml is sometimes required. Some anaesthetists prefer to use thiopentone or hexobarbitone for induction of anaesthesia. The authors recommend the drip method for induction of anaesthesia especially in asthmatic patients and to quieten patients during spinal analgesia.

local trauma. Finally, acute attacks may occur, which may send the patient to the operating theatre as an acute abdominal emergency. There may be a history of passing red urine and of paralysis following previous operations of skin lesions, etc. If thiopentone is used in these cases a lower motor neurone paralysis is likely to develop afterwards. Patients suffering from this disorder of metabolism are said to tolerate ether quite well and for pre and post operative sedation opiates, chloral hydrate, and paraldehyde are said to have no ill effects.

Bemegride (megimide) would appear to be a useful antidote to the narcotic and respiratory depressant actions of thiopentone⁵⁴ and a safe means of terminating thiobarbiturate anaesthesia. The dosage is 25 mg intravenously repeated two or three times at short intervals if necessary. This agent reverses the E E G patterns of deep depression due to barbiturates and thiobarbiturates, but not those caused by non barbiturate sedatives. This reversal is greater than that produced by other analeptics.

It is well known that intravenous injection of pethidine will produce a transient weal should the vein be obstructed or superficial. This phenomenon is probably due to the liberation of histamine in the skin overlying the vein following permeation of the vein wall by pethidine. It is recommended⁴ that the drug should be used in a diluted form for example 1 per cent and in those patients who show a tendency to thrombosis it should be diluted with procaine 0.25 per cent which releases spasm in the vein and is an anti histaminic drug. Substitutes for pethidine have been sought to supplement gas oxygen thiopentone anaesthesia and one of these is Nisentil (alphaprodine).

Nisentil (alphaprodine). This drug 1-3 diamethyl-4 phenol-4 propionyxy piperidine was first synthesized by Lee in 1947. It is similar both chemically and pharmacologically to pethidine but acts for a shorter time and shows its effects more quickly. It can be used to replace pethidine in the thiopentone gas oxygen relaxant intravenous technique using either repeated fractional doses of 5 to 10 mg or as a drip of 0.01 per cent solution. Success has been claimed⁴ for a method which uses nisentil together with suxa methonium in a continuous drip of 0.01 per cent and 0.1 per cent respectively. One must however question the wisdom of using two drugs of different type in a fixed proportion.

It has been successfully used to relieve the pain of first stage labour and the action of a dose of 40 to 60 mg lasts about 45 minutes. Untoward effects are similar to those of pethidine and

Intravenous Procaine This does not seem to have gained many habitual users in recent years. The vaso dilatation that it causes increases the surgeon's difficulties by accentuating oozing. In dogs it would seem to increase the excretion of sodium when added to intravenous saline, an effect possibly accounted for by diminished renal tubular reabsorption of sodium. Procaine and morphine too, inhibit water diuresis⁶⁵ although an increase of diuresis following the administration of normal saline containing procaine is reported⁶⁶.

New Apparatus A simple intravenous drip adaptor has been described by Green³¹. This enables drugs to be injected into the drip close to the needle with the arm at the side of the patient.

A similar piece of apparatus has been described by Lee. This consists of a length of neoplex plastic tubing into one end of which fits a transfusion needle minus its hub and into the other end of which is inserted another needle attached to a 3 way II D tap. A 20-ml syringe containing saline is used to wash through the drug injected into the third arm of the 3 way tap. By the use of these pieces of apparatus more room is given to the surgical assistants and the patient does not run the risk of brachial plexus paralysis which sometimes occurs when the arm is abducted from the side.

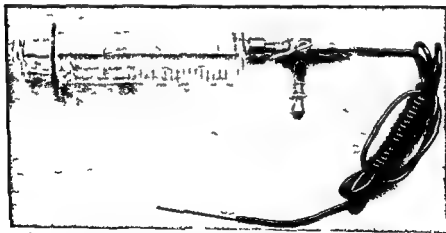


FIG. 51 The Lee television apparatus for intravenous injections with the patient's arm at his side (Southend Hospital Department of Photography)

In this age of electronics it is not surprising that automatic electroencephalographic control of anaesthesia is in vogue in certain centres³. As a logical sequence in the development of automatically controlled anaesthesia with ether is the extension of clinical investigation to include thiopentone anaesthesia and an apparatus has been

It may be used as a basal hypnotic together with local or topical analgesia for endoscopy, and is useful for oral and facio maxillary operations. It works well also in status asthmaticus and in some cases of tetanus. Post operative sleep lasts rather longer than after comparable doses of thiopentone. The main drawback is the thrombophlebitis which is sometimes seen. It comes on within the first week after operation and occurs in about 6 per cent. of cases. More dilute solutions are not practicable as too large volumes of fluid would thereby be required. This high incidence of thrombophlebitis may be a sound reason for avoiding the technique.

The effects of sodium succinate on thiopentone anaesthesia is discussed from time to time. In the opinion of Gairman *et al*,²⁸ it seems that the analeptic effects of this salt and its derivatives reported a decade ago do not in fact occur. These workers state that on the contrary it has a potentiating effect on thiopentone anaesthesia. This work is confirmed by Larsen and his colleagues.⁹ On the other hand, *in vitro* experiments³⁰ show that pentobarbitone depresses the oxygen consumption of brain slices and that this depression is prevented by sodium succinate—a clear difference between *in vitro* results and clinical anaesthetic experience.

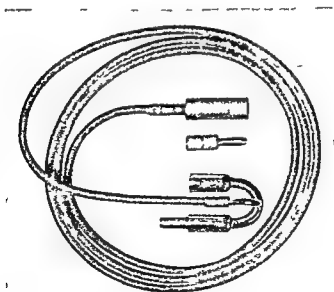


FIG 50 Apparatus for intravenous injection (Green *Brit J Anaesth*)

- 8 DUNDEE, J W 1955 *Anaesthesia* 10 193 (April)
- 9 MUSHIN W W *et al* 1955 *Brit J Anaesth* 27 374 (Aug)
- 10 JOLLY C P and LEDERER J 1955 *Anaesthesia* 10 292 (July)
- 11 TOVELL, R M *et al* 1955 *Anesthesiology* 16 910 (Nov)
- 12 DUNDEE J W and RIDING J E. 1955 *Brit J Anaesth* 27 381 (Aug)
- 13 LUND P C 1954 *Anesth & Analges* 33, 86 (March)
- 14 PHILLIPS H S 1953 *Anesth & Analges*, 32 56 (Jan)
- 15 BRYCE SMITH R and HINGSON R A 1954 *Anesthesiology* 15, 743 (July)
- 16 HARRISON GWENDOLINE, 1953 *Brit J Anaesth* 25 204 (July)
- 17 MYERS F H *et al.*, 1954 *Anesthesiology* 15 146 (March)
- 18 DUNDEE, J W and ROBERTS R K. 1954 *Anesthesiology* 15 333 (July)
- 19 SESSIONS G W *et al* 1955 *Anesthesiology* 16 235 (March)
- 20 DUNDEE, J W 1955 *Brit J Anaesth* 27 14 (Jan)
- 21 DUNDEE, J W and ANNIS D 1955 *Brit J Anaesth* 27 144 (March)
- 22 DUNDEE, J W., 1954 *Brit J Anaesth* 24 164 (May)
- 23 FIELDMAN E J *et al* 1955 *Anesthesiology*, 16 473 (July)
- 24 ARGENT D E., and DINWICK O P 1954 *Brit J Anaesth* 26 260 (July)
- 25 SIKER E S, FOLDES F F *et al* 1954 *Brit J Anaesth* 26 405 (Nov)
- 26 SCOTT D C 1955 *Brit J Anaesth* 27 393 (Aug)
- 27 DWYER, C S *et al* 1953 *Anesthesiology* 14 291 (May)
- 28 GARMAN N J., *et al* 1954 *Anesthesiology* 15 122 (March)
- 29 LARSEN E *et al* 1954 *Anesthesiology* 15 537 (Sept)
- 30 LARSEN F 1955 *Anesthesiology* 16 239 (March)
- 31 GREEN R. 1954 *Brit J Anaesth*, 26 287 (July)
- 32 KIERSEY D K. and FAULCONER A *et al* 1954 *Anesthesiology* 15 356 (July)
- 33 STUART P 1955 *Brit med J* 2 1308 (Nov 26)
- 34 WEISE, H and KOSS F H 1954 *Dtsch med Wschr* 79 601
- 35 NOBLE P 1955 *Lancet*, 1 797 (April 16)
- 36 EVANS P T 1955 *Lancet* 1 291 (Jan 2)
- 37 ZUCK D 1955 *Lancet* 2 1277 (Dec 17)
- 38 BOTTOMS R. W H 1954 *Lancet* 1 1167
- 39 HOWLAND W S *et al* 1956 *Anesthesiology* 17 1 (Jan)
- 40 BELLVILLE, J WELDON *et al* 1956 *Brit J Anaesth* 28 50 (Jan)
- 41 TAYLOR, N and SHEARER W M 1956 *Brit J Anaesth* 28 67 (Jan)
- 42 MILLER E *et al* 1936 *J Amer chem Soc* 58 1090
- 43 SELYE, H., 1941 *J Immunol* 41 259
- 44 LAUBACH G D PAN S Y and RUDEL, H W 1955 *Science* 122 78
- 45 MURPHY F J *et al* 1955 *J Amer med Ass* 158 1412
- 46 GALLEY A H and ROOMS M 1956 *Lancet* 1 990 (June 23)
- 47 LERMAN L H 1956 *Brit med J* 2 129 (July 7)
- 48 TAYLOR, N and SHEARER W M 1956 *Brit J Anaesth* 28 67
- 49 *Lancet* Annotation 1 1002 (June 23)
- 50 HARBORD R P and WILD W M 1956 *Proc R Soc Med* 49 487 (July)
- 51 REIFFERSCHIED M and DEITMANN K 1954 *Dtsch med Wschr* 79 638
- 52 BOONE, J D *et al* 1956 *Anesthesiology* 17 284
- 53 FROMEK A and PISA Z. 1956 *Brit J Anaesth* 28 366 (Aug)
- 54 BENTEL, H *et al* 1956 *Med Proc* 2 198 (April)
- 55 HALINT P., and HAJDU A 1956 *Lancet* 2 466 (Sept 1)
- 56 PRONINA, N N 1955 *Bull exp Biol Med U.S.S.R* 5 12
- 57 DUNDEE, J W 1956 *Thiopentone and other Thiobarbiturates* Edinburgh E. & S Livingstone
- 58 YOUNG M S 1956 *Proc R Soc Med* 49 735 (Oct)

designed which performs this function. Its behaviour is not influenced by the simultaneous injection of tubocurarine. Is automation peeping round the door of the operating theatre?

An electrical drip recorder has been described³⁶ which enables a nurse in a darkened room to observe the rate of transfusion at a glance. A modification³⁷ consists of a specially sensitive spring loaded switch which illuminates a steady warning light when the

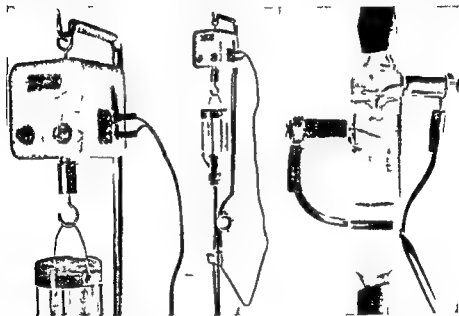


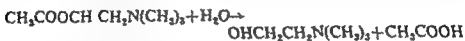
FIG 52 The Frankis Evans drip recorder modified by Zuck (Lancet)

weight of the bottle and its contents falls to a predetermined level. The warning device can be used alone if required and it is set in action simply by hanging it from a support and suspending the bottle from the hook underneath. It uses a high tension battery and a neon bulb so current consumption is negligible. Another drip alarm has been described by Bottoms³⁸

References

- 1 BROWN A K 1953 *Brit J Anæsth* 8 26 (Jan)
- 2 MEALS W G 1948 *Anesthesiology* 9 644 (Nov)
- 3 EVANS F T and GRAY P W S 1953 *Anæsthesia* 8 104 (April)
- 4 LANCASTER F M and LEVIN J 1956 *Brit med J* 1 381 (Feb 18)
- 5 BURNS T H S 1954 *Anæsthesia* 9 45 (Jan)
- 6 CLIVE LOW S G DE and GRAY P W S 1954 *Anæsthesia* 9 96 (April)
- 7 DUNDEE J W and RIDING J E 1955 *Anæsthesia* 10 55 (Jan)

myoneural junction, acetylcholine is released from the nerve ending and depolarizes (or renders electrically negative) the muscle end plate, i.e. it causes an end plate potential. This current of depolarization excites the adjoining muscle fibre (or evokes an action potential) and propagation of this excitation wave along the membrane of the muscle fibre causes contraction of the contractile elements. In the meantime the released acetylcholine has been hydrolysed by the enzyme cholinesterase always present in tissues into relatively inactive choline and acetic acid



so that when the muscle fibre emerges from its refractory period, it will not become excited again until a fresh nerve impulse arrives

If curare or a similar drug is given intravenously, no muscular contraction follows motor nerve stimulation. It is supposed that these drugs are sufficiently like acetylcholine to have some affinity for its receptor sites but sufficiently unlike it to be unable to initiate the process of depolarization. They thus compete for receptor sites with acetylcholine and reduce the activity of a given quantity of the latter compound. They are therefore known as competitive neuromuscular blockers or non-depolarizers.

A second type of muscle relaxant is known and these drugs resemble acetylcholine so closely that they not only have an affinity for the same receptor sites but may initiate impulses by causing depolarization. This is often seen in fibrillary twitching of the neck muscles soon after the intravenous injection of the relaxant. It has been proved that the subsequent block is due to depolarization as it can be removed electrically by applying an anode to the end plate. These drugs have therefore been termed depolarizing blockers.

Although it is useful to make a rigid subdivision of blockers as described above some drugs are known (such as mytolon) which appear to possess both actions, while the occasional prolonged action of suxamethonium is thought to be sometimes due to its depolarizing effect giving place to a non-depolarizing one, i.e. it may be a mixed blocking agent.

Antidotes to competitive relaxants exist in the anticholinesterases the one generally used being neostigmine. Their action is mainly that of neutralizing cholinesterase so that the acetylcholine can build up sufficiently to break through the barrier to the receptor sites.²⁵ Neostigmine can also however stimulate muscle end plates directly.²⁶ Antidotes are considered in detail later in this chapter.

CHAPTER 7

MUSCLE RELAXANTS

MUSCLE tone is probably due to a slow asynchronous discharge of impulses from anterior horn cells producing a partial tetanus. Abolition of this tone or muscular relaxation can be brought about during anaesthesia in three ways

(1) By profound general narcosis This is a central effect probably associated with depression of the anterior horn cells in the spinal cord

(2) By isolating muscles or groups of muscles from all impulses by blocking their nerve supply by local analgesics

(3) By blocking impulses to all muscles at the myoneural junctions by certain drugs given intravenously This is a peripheral effect and the drugs concerned are known as specific muscle relaxants

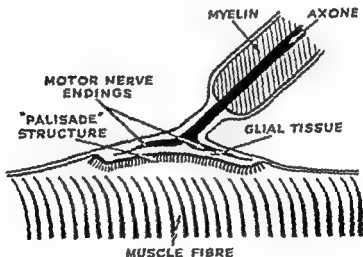


FIG 53 Diagram of myoneural junction (Paton *Anaesthesia*)

Theory of Action

The current theory of normal striped muscle contraction is that when an impulse travelling along a motor nerve arrives at the

mixtures of alkaloids but even as early as 1858-1861, Spencer Wells advocated the use of "woorari" for the treatment of tetanic spasms and in 1878 definite success in this field was claimed ⁵

At last in 1935 a pure alkaloid was isolated in England by H. King of The National Institute of Medical Research and named d-tubocurarine chloride ⁶. This was found to be a quaternary ammonium compound (It might be noted in passing that the corresponding l alkaloid has only $\frac{1}{2}$ of the physiological activity)

Since that date progress has been rapid. In August, 1939, the drug was first used in London at the Middlesex Hospital to lessen the risk of trauma from electro-convulsive therapy in mental patients ⁷. It was then tried out with great success as a muscle relaxant during light general anaesthesia the first large series of cases being described in Canada by Griffith and Johnson ⁸ and in England by Gray and Halton ⁹.

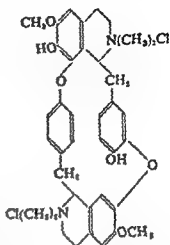
Action (1) On Nervous and Muscular Systems Tubocurarine has negligible lipid solubility so that it probably does not penetrate cells ¹⁰. When curare is injected intravenously it takes about 90 seconds to act. This is greater than the "arm brain circulation time" and would in itself suggest a peripheral rather than a central effect. The mode of action of curare in paralysing striped muscle has already been discussed.

All voluntary muscles are affected by large doses of curare, becoming paralysed and flaccid but they are not all acted upon to the same extent for example those of the eyes are particularly susceptible.

Curare also paralyses sympathetic ganglia and it is possibly this action which minimizes traumatic shock even if a severe stimulus is inflicted on a lightly anaesthetized patient.

When used in very small dosage to conscious patients, it is said that curare and some other relaxants have a lissive effect. This means that they diminish muscle spasm without impairing voluntary muscle power. This action may be exploited in the treatment of spastic states, often combined with physiotherapy ¹¹.

In ordinary dosage curare has no effect on the cerebral cortex ¹² and as no anaesthetic or analgesic action is present a light general



The relaxants in common use can be classified as in the following table —

Type of Relaxant	Duration of effect of single dose		
	Long (30-60 mins)	Medium (15-30 mins)	Short (2-5 mins)
Non depolarizing (competitive) blockers	d tubocurarine chloride (tubarine) d methylether of tubocurarine chloride (mecosium) bromide (diamethane) iodide (D M E.) laudarium methyl sulphate (laudolissum)	gallamine triethiodide (flaxedil)	
Depolarizing blockers		decamethonium (C. 10) dibromide (sincurone) diiodide (eulissin)	suxamethonium (succinyl dicholine) chloride (scoline) bromide (brevidil M powder) iodide (celocurine) suxethonium chloride bromide (brevidil E. powder) iodide (LS 362 Bovet)

The individual drugs must now be considered separately

CURARE

History Curare also known as wourari wourali urari and uririrery was first described as an arrow poison by Sir Walter Raleigh to Queen Elizabeth on his return to England from his voyage to the Orinoco in 1584 *

In 1800 Humboldt found that certain Indian tribes in South America were smearing on their arrow heads and blow pipe darts a syrup obtained from a creeper which was later named *strychnos toxifera*

In 1812 Charles Waterton in his book *Wanderings in South America* gave a detailed description of the paralysis which ensued when an animal was hit by an arrow poisoned with curare as the concoction was now called and in 1814 he and Brodie stated that the paralysis was probably due to a toxic action on the neuromuscular mechanism

The first scientific research work on the substance was carried out about fifty years later by the French physiologist Claude Bernard³ who in his classical series of experiments proved that the poison acted on the myoneural junction

Therapeutic use of curare in the human subject was then held up for a long time owing to the toxic effects notably bronchospasm⁴ which occurred fairly frequently when using unknown and differing

a valve which tends to obstruct the passage of fluids, from the pharynx to the œsophagus but not in the reverse direction ⁷⁵

(4) *On Cardiovascular System* In normal dosage curare has little effect on the blood pressure of anesthetized patients but slight falls are not uncommon (86 per cent in one series) The cause is probably sympathetic ganglia paralysis or histamine release or both factors In experimental arterial injection this effect is easily demonstrated ²¹ On the other hand, rises in blood pressure occasionally occur and may be spectacular ²⁰ The original suggestion that the reason is increased carbon dioxide tension from depressed respiration is unlikely to be true as forced hyperventilation with an absorber does not bring the pressure down to its original level No clinical changes in the cardiac rhythm and no typical electro cardiographic changes have been noted Curare does not appear to afford any protection against vagal inhibition of the heart during anesthesia ²² The injection of large doses of the alkaloid into the circuit of the heart lung preparation in the dog has no effect on cardiac output, arterial pressure or coronary flow It does however cause a slight fall in venous pressure which would indicate an improvement of the cardiac condition ²³

Increased bleeding from wounds is occasionally observed in curarized patients This has been ascribed to the release of heparin

Elimination Curare is eliminated fairly rapidly, partly by destruction in the liver and partly by being passed unchanged in the urine It seems to be established, however, that patients with gross liver damage usually show a resistance to the normal effects of curare ²⁴ One explanation of this phenomenon is that in hepatic damage the serum pseudo cholinesterase is lowered and this might lead to an increased sensitivity to acetylcholine at the end plates ²⁵ In ordinary dosage, curare does not appear to pass the placental barrier to any significant extent and thus the foetus is unaffected by it

Preparations and Dosage Crude curare is classified quite unscientifically by the containers in which it is packed Thus three varieties are recognized (1) Calabash or gourd curare (2) pot or jar curare and (3) tube curare which is placed in bamboo tubes It has been said that these varieties of crude curare contain differing proportions of curarine and allied alkaloids such as protocurarine, protocurine and curine As however the properties and even the identities of these substances are uncertain it is unprofitable to pursue the subject further

There are several therapeutic preparations available at the present time

narcosis must be maintained. In this connection a case has been reported of a curarized patient regaining consciousness and sensitivity to pain during the course of an abdominal operation but being quite unable to indicate the fact.¹² In very large doses, curare has a central as well as a peripheral action and consciousness may be lost.

(2) *On Respiratory System* It has already been stated that curare does not affect all muscles to the same extent. If sufficient dosage is given for abdominal relaxation the intercostal muscles will almost certainly be paralysed and the breathing will become diaphragmatic. Direct recordings of the effect of a given dose of curare on the contractions of the diaphragm and of a voluntary muscle have shown that the former is less susceptible than the latter whether stimulated naturally or artificially through the phrenic nerve. The 'therapeutic index' of curare,¹⁴ i.e. the ratio of the dose required to arrest breathing to that needed to abolish muscle tone has been estimated at about 1.4. It is therefore obvious that it may prove impossible to secure complete relaxation with efficient natural breathing and if curare is used the anaesthetist must be prepared to use assisted or controlled respiration.

The opinion is now held by some anaesthetists that complete apnoea is not only innocuous but actually desirable.¹⁵ It might perhaps be pointed out that controlled respiration carried out either by intermittent hand pressure on a reservoir bag or by elaborate mechanical respiration by no means simulates natural breathing in the differential pressures obtained and under certain conditions may prove detrimental.

Bronchospasm although rare does occur even with pure preparations of tubocurarine. It is probably due to histamine release¹⁷ and in the authors' opinion is practically never encountered unless thiopentone is being administered at the same time or has recently been injected and unless anaesthesia is too light.

(3) *On Gastro intestinal Tract* Although it is usually stated that curare has no effect on smooth muscle it would seem likely that since the intestinal musculature contracts by acetylcholine action it would be affected by the drug. In most animals curare inhibits intestinal peristalsis¹⁸ but in man the effect is variable. The stomach and gut are usually quiescent but occasionally they exhibit increased movements.⁹ The intestines are rarely contracted as often seen during spinal block. Contrary to previous belief the cardiac sphincter does not seem to be relaxed with full doses of myoneural blockers. The cricopharyngeal sphincter is said to be converted into

facilitate controlled respiration and to avoid the cough reflex which may be so troublesome when the surgeon is working near the hilum of the lung. If short periods of relaxation only are required, e.g. for bronchoscopy or in electro convulsive therapy, it is preferable to substitute a shorter acting agent such as gallamine or suxamethonium for curare.

Contra-indication Curare should be avoided in patients suffering from myasthenia gravis except for the minute test dose sometimes used for diagnosing this condition.

Technique of Administration Curare has no effect by mouth and is usually given intravenously, but when this is impossible the intramedullary or intramuscular route can be used. For a very prolonged effect, e.g. for controlling the spasms of tetanus, curare can be given intramuscularly dissolved in wax and peanut oil.

The original and still sound method of giving curare for the average long abdominal operation is to establish the patient in a steady but light plane of general narcosis and then to give an initial intravenous dose of (say) 10 mg. If relaxation is insufficient in three minutes a supplementary dose of 5 mg is given. When the respiratory exchange becomes insufficient, assisted or fully controlled respiration is instituted. No curare should be given within 30 minutes of the completion of the operation and if the muscles tighten during peritoneal closure a small dose of intravenous thiopentone will always relax them temporarily. In the writer's opinion this is preferable to using a short acting relaxant such as suxamethonium as sometimes prolonged apnoea follows this sequence the reason for which is not entirely clear. In many cases an intravenous drip will be set up and the injection can conveniently be made into the rubber tubing as near the needle as possible. Otherwise a Gordh's needle (q.v.) or one of its modifications is satisfactory. As a general rule endotracheal anaesthesia is desirable with curare not so much for fear of laryngospasm or bronchospasm but as a safeguard against gastric regurgitation. A cuff or pack is, of course, essential for this purpose.

It is not always easy to decide when an additional dose of curare is indicated and it is worth noting that an increase in the tidal respiratory volume generally occurs just before the return of muscle tone.

An alternative technique which has become popular is to give a preliminary dose of 5 mg curare to the conscious patient. This should produce no obvious effect beyond a mild ptosis but occasionally patients are met with who appear to have a true idiosyncrasy

(a) "Tubarine" (B W) is most commonly used in Great Britain and is claimed to be identical with the pure alkaloid isolated by King. It replaced the former product "curarine chloride" (B W) which was a white powder which had to be dissolved and sterilized. Tubarine is a sterile solution containing 10 mg per ml and is supplied in 1.5 ml ampoules and 5 ml rubber capped bottles. The ordinary preparation forms an insoluble precipitate when mixed with thiopentone but "tubarine miscible" is also available for mixed injections.

(b) d-tubocurarine chloride (Duncan) is a solution of the same potency as tubarine. It contains glycerine and alcohol and can be mixed with thiopentone.

(c) "Myostatine" is a similar preparation to tubarine.

(d) "Intocostin" (Squibb) is an American preparation of a yellow sterile solution containing 20 mg of 'curare extract' per ml with 0.5 per cent chlorbutanol added as a preservative. It is supplied in rubber capped bottles of 5 ml and 10 ml capacity and is a purified extract from the plant *chondodendron tomentosum*. As this is not a pure solution of d-tubocurarine chloride its action is more variable than that of the British product⁸ and the dosage is higher, 2 to 3 ml (40 to 60 mg) being an average initial dose, i.e. it has about one fourth the potency of tubarine.¹¹

It is unfortunate that no international agreement has yet been reached to standardize the potency of curare preparations and it is at present imperative to distinguish between them.

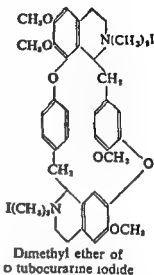
All curare solutions keep best in a refrigerator but if they become cloudy they should be discarded.

(e) "Tubodil" is a suspension of tubocurarine chloride in a mixture of peanut oil, oxysterol derivatives and beeswax in a concentration of 25 mg of the alkaloid in 1 ml. A dose of from 1 to 3 ml intramuscularly is said to give relief for up to 24 hours from the pain of muscle spasm from any cause.

Indications. Curare can be given in most cases where complete muscular relaxation is required for some time. The most obvious scope for its use is in upper abdominal or thoraco abdominal surgery where there is a remarkable absence of shock from severe trauma such as peritoneal traction. A possible reason for this has already been mentioned. Curare appears to cross the placental barrier from mother to foetus very slowly if at all¹⁰ although it has been shown in animals to pass in the reverse direction.⁷ There therefore seems no reason to withhold its use in obstetrics e.g. in Caesarean section. Curare is also useful during thoracotomies to

paralysis and histamine release are no greater in spite of the increased potency

DME is available in 3 ml ampoules containing 6 mg of the drug. After considerable clinical use it is the general opinion that DME has a slightly shorter period of causing diaphragmatic paralysis than curare and that the inflation of collapsed lungs and lobes is slightly easier during thoracotomy this effect being probably due to less histamine release with correspondingly less bronchiole constriction. It would seem therefore that DME may have a slight advantage over curare but owing to the fact that the complicated molecule has four optical isomers it is difficult to be certain that all samples are identical.³³



DI METHYL ETHER OF D TUBOCURARINE BROMIDE

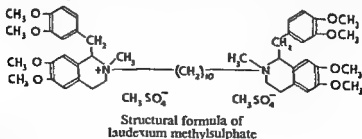
This preparation also known as diamethine has a similar action to the iodide and is supplied in 1.5 ml ampoules containing 6 mg and in rubber capped bottles of 20 mg

DI METHYL ETHER OF H TUBOCURARINE CHLORIDE

This is a similar compound also known as mecostrin (kondrocurare)

LAUDEXIUM METHYLSULPHATE

This drug is one of the heterocyclic decamethylene bis quaternary ammonium compounds and is also known as laudolissin and as



"compound 20" It is a competitive blocker and is consequently antagonized by neostigmine but not to the same extent as is H tubocurarine. Its potency is about half that of tubocurarine but

to the drug although not suffering from myasthenia gravis. Should a normal response occur, a further 10 mg. is given followed immediately by a suitable dose of thiopentone. Complete relaxation unconsciousness and depression of respiration will follow. An artificial airway or tracheal tube is then passed and anaesthesia maintained with the desired method accompanied at first by assisted or controlled respiration.²⁸ This technique colloquially termed a 'crash induction' is spectacular and rapid, but is not devoid of risk. In the first place it is possible for the needle to slip out of the vein before the thiopentone has been given, and if difficulty in reinsertion is experienced the patient will have the unpleasant sensation of becoming curarized without losing consciousness. Secondly the anaesthetist must be sure that the stomach is empty or regurgitation may occur before tracheal intubation can be carried out. This danger is of course, aggravated in such conditions as acute intestinal obstruction²⁹ unless the induction of anaesthesia is carried out with the patient in a steep head up slope.

The nature of the general narcosis accompanying curarization varies to some extent with the operation. The author prefers nitrous oxide and oxygen (about 70/30) with minute additions of ether trichlorethylene or cyclopropane. If a non volatile agent is required pethidine seems preferable to thiopentone except for closing the abdomen at the end of the operation. If the latter drug is employed for long there is a tendency towards jerky respiration and hiccough and the blood pressure curve seems to show greater deviation from normal. Whatever method is used adequate oxygenation and removal of carbon dioxide are of paramount importance.

After-effects The only after effects attributable to curare so far reported are paresis of some of the ocular muscles which may persist for a few days.¹² Further information on curare will be found under abdominal surgery.

DI METHYL ETHER OF D TUBOCURARINE IODIDE

The preparation of the di methyl ether derivative of tubocurarine chloride (D M E) was first described by H. King in 1935³⁰ and its pharmacology has now been worked out in animals³¹ and in man.³ It has been established that D M E acts in the same way as the parent drug and is antagonized by the anticholinesterases such as neostigmine.

The chief differences between the two preparations are that D M E is 2 to 2½ times as potent and that sympathetic ganglia

For this reason gallamine is useful for operations lasting for 20 to 30 minutes which require muscular relaxation for most of the time. The drug appears to have less effect than curare on paralyzing sympathetic ganglia but it tends to cause tachycardia due to a vagolytic action. It should therefore protect the heart to some extent from the effects of vagal activity during anaesthesia. In the presence of carbon dioxide retention, however, it is said that gallamine can cause ventricular arrhythmia. It is antagonized by neostigmine and should not be used on patients suffering from myasthenia gravis or from greatly reduced renal function as elimination occurs by the kidneys. Gallamine appears to pass the placental barrier with some ease and should therefore not be used in obstetrics.

DECAMETHONIUM IODIDE

Decamethonium ("C 10") is bistrimethylammonium decane $[N(CH_3)_3(CH_2)_{10}N(CH_3)_3]2I$. The dibromide is also known as syncurine and the diiodide as eulissin. Both salts are supplied as solutions containing 2 mg per ml. Its effects were studied by the Anaesthetics Committee of the Royal Society of Medicine and Medical Research Council and it was found to be a stable non-irritating solution miscible with thiopentone. Its potency (by weight) is about five times that of d-tubocurarine. 3 mg usually producing a short period of complete muscular relaxation in the fit adult.³⁸

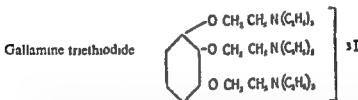
Unlike curare decamethonium is a depolarizing relaxant (see earlier) and consequently the anticholinesterases such as neostigmine have little antagonizing action although they are, in fact, used by some workers. A further difference is that patients with myasthenia gravis have an *increased* tolerance for decamethonium³⁹ although severe cases apparently are hypersensitive to it. In the latter circumstances the block produced is a non-depolarizing one so that decamethonium can evoke a dual response.⁷⁶ It was at first thought that lower members of the same series (e.g. pentamethonium and hexamethonium) could be used as antidotes to decamethonium. It was soon realized however that in effective doses these drugs caused profound falls in blood pressure from their action of blocking autonomic ganglia and were useless for the purpose (see Chapter 10).

Decamethonium has about the same length of action as gallamine and can be used for the same purposes. The authors have not found it as reliable while undesirable side effects are common, e.g. bradycardia and fall in blood pressure.⁴⁰

its duration of action is slightly longer. On the other hand a longer time elapses between an intravenous injection and the full relaxant effect. It cannot be mixed in the same solution with either thiopentone, atropine, gallamine or pethidine³¹. It has no obvious side effects except the possible liberation of histamine and like tubocurarine affords no protection against vagal inhibition of the heart³². Laudexium was synthesized by Collier and Taylor in 1951 and is usually supplied in 1.5 ml ampoules containing 30 mg.

GALLAMINE TRIETHIODIDE

Gallamine triethiodide (flaxedil, sincurarine and "R P.3697") is tri (β diethylaminoethoxy) benzene triethyliodide, and was first synthesized in France in 1947³³. It is a white amorphous powder with a melting point of 145–150° C and a molecular weight of 891. It is supplied in a 4 per cent solution in water for intravenous injection (i.e. 40 mg per ml). This is miscible with thiopentone.



The first long series of cases with gallamine was reported in this country³⁴ in 1949 and it has now been shown that in general it has a curare like effect but is about one fifth as potent, 80 mg being equivalent to about 15 mg curare. The duration of action is about half that of curare as is well shown in the accompanying graph³⁷.

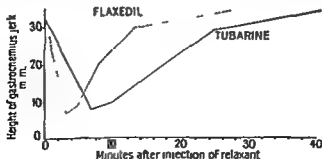


FIG. 54 Chart comparing duration of action of gallamine (flaxedil) and curare (Doughty and Wylie *Proc R Soc Med*)

For this reason gallamine is useful for operations lasting for 20 to 30 minutes which require muscular relaxation for most of the time. The drug appears to have less effect than curare on paralyzing sympathetic ganglia but it tends to cause tachycardia due to a vagolytic action. It should therefore protect the heart to some extent from the effects of vagal activity during anaesthesia. In the presence of carbon dioxide retention, however, it is said that gallamine can cause ventricular arrhythmia.² It is antagonized by neostigmine and should not be used on patients suffering from myasthenia gravis or from greatly reduced renal function as elimination occurs by the kidneys. Gallamine appears to pass the placental barrier with some ease and should therefore not be used in obstetrics.

DECAMETHONIUM IODIDE

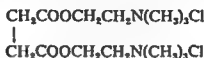
Decamethonium ("C 10") is bis(trimethylammonium) decane $[N(CH_3)_3(CH_2)_{10}N(CH_3)_3]2I$. The dibromide is also known as syncurine and the diiodide as culissin. Both salts are supplied as solutions containing 2 mg per ml. Its effects were studied by the Anaesthetics Committee of the Royal Society of Medicine and Medical Research Council and it was found to be a stable non-irritating solution miscible with thiopentone. Its potency (by weight) is about five times that of d-tubocurarine, 3 mg usually producing a short period of complete muscular relaxation in the fit adult.³⁸

Unlike curare, decamethonium is a depolarizing relaxant [see earlier] and consequently the anticholinesterases such as neostigmine, have little antagonizing action although they are, in fact, used by some workers. A further difference is that patients with myasthenia gravis have an *increased* tolerance for decamethonium,³⁹ although severe cases apparently are hypersensitive to it. In the latter circumstances the block produced is a non-depolarizing one so that decamethonium can evoke a dual response.⁷⁶ It was at first thought that lower members of the same series (e.g. pentamethonium and hexamethonium) could be used as antidotes to decamethonium. It was soon realized, however, that in effective doses these drugs caused profound falls in blood pressure from their action of blocking autonomic ganglia and were useless for the purpose (see Chapter 10).

Decamethonium has about the same length of action as gallamine and can be used for the same purposes. The authors have not found it as reliable while undesirable side effects are common, e.g. bradycardia and fall in blood pressure.⁴⁰

SUXAMETHONIUM CHLORIDE

[Succinylcholine chloride, scoline, suxamyl, anectine, lystenon pantolax, brevidil M solution] Suxamethonium chloride is the most generally used short acting relaxant and has the structural formula



The solution contains 50 mg per ml and the dose varies from 2 ml in a large muscular adult to 0.5 ml in a child (or about 0.5 mg per lb for intubation or other short procedures)

The action of suxamethonium is that of depolarization and it can be regarded as a short acting decamethonium (qv). Its short effect is due to the fact that it is hydrolysed by pseudo-cholinesterase instead of being excreted by the kidneys as is C 10. Neostigmine should therefore *not* be used as an antidote.⁵⁷

Although the drug is hydrolysed by alkalis it can be mixed with thiopentone if the mixture is injected immediately. It is, however, a poor technique as the preliminary wave of muscular contraction is painful and may occur before consciousness is lost. Incidentally generalized muscle pains or 'sore throat' due to stiffness of the pharyngeal muscles may persist for several days afterwards in ambulant patients. Those confined to bed apparently complain less of this complication.⁴¹ A better method is to start with a sleep-dose of thiopentone and then to inject suxamethonium. The lungs should be rhythmically inflated with oxygen as spontaneous respiration will probably cease for some minutes. It is however essential to remember that suxamethonium may permit the passive regurgitation of stomach contents if the patient is lying flat. Much caution should therefore be exercised when using the drug on outpatients or when there is any reason to believe that the stomach may not be quite empty. Since its introduction it has been used with great success for such short procedures as intubation examinations under anaesthesia, ECT⁴ and manipulations.⁴³ Suxamethonium is thought to cause less cardiovascular changes during laryngeal intubation than other relaxants but it may have a muscarine like action causing bradycardia. This is abolished by a preliminary injection of atropine.²²

In a few patients the action of suxamethonium is considerably prolonged over the usual 3 to 4 minutes.⁴⁴ The most likely explanation of this phenomenon is that such patients have an abnormally low level of plasma pseudo cholinesterase and thus the suxamethonium is only slowly converted into succinic acid and choline.⁴⁵

It has been observed that most of such patients are ill clinically and may be suffering from severe anaemia, liver disease, starvation, thyrotoxicosis or electrolyte imbalance. It is thought that sometimes the depolarizing block is replaced by a non depolarizing one [dual response] and this may prolong the effect⁴⁴. Intravenous procaine also prolongs the action of suxamethonium probably because both are hydrolysed by pseudo cholinesterase⁴⁷ (which may be identical with the enzyme known as procaine esterase). Other causes of prolonged apnoea include overdose of the relaxant, prolonged administration of a dilute solution (see next paragraph), reflex laryngeal spasm and too much or too little carbon dioxide in the blood stream.

The drug has also been given in diluted form⁴⁸ (e.g. 0.15 per cent or 750 mg in 500 ml normal saline)⁴⁹ as an intravenous drip during prolonged operations with the idea of maintaining a constant degree of relaxation. There are several drawbacks to this technique. In the first place any method of anaesthesia which depends on the correct functioning of a drip infusion tends to be unreliable. Secondly in order to obtain a sufficient degree of relaxation it may be necessary to overload the patient's circulation with fluid. Lastly a few patients exhibit prolonged apnoea after cessation of the drip. This is thought to be due to an accumulation of succinyl monocholine⁵⁰ which is an intermediate product in the breakdown of suxamethonium. In vitro at any rate it has a weaker but much more prolonged effect than the parent drug. A prolonged drip infusion of suxamethonium has been used with success in the treatment of tetanic spasms⁵¹.

SHORT ACTING DEPOLARIZING RELAXANTS

<i>Active Cation</i>	<i>Inactive Anion</i>	<i>Synonyms</i>
suxamethonium (succinylcholine)	chloride	anectine brevital M solution lysthenon pantolax
	bromide	scoline brevital M powder
	iodide	celocurine curacit s c i
suxethonium	chloride	brevital E powder I S 362 Bovet
	bromide	
	iodide	

The chemical relationship of most of the short acting relaxants which have been used in anaesthesia are summarized in the table on p 113⁵ but in practice suxamethonium chloride is almost always used in this country

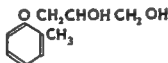
It should be noted that the methonium cation is about $2\frac{1}{2}$ times as potent as the ethonium one. Scientifically the dosage should really be calculated in milligrams of active cation but in practice this has proved to be too complicated. As an example 50 mg suxamethonium chloride contain 40 mg active cation whereas 60 mg of the bromide are required

MISCELLANEOUS RELAXANTS

There remain a few muscle relaxants which for one reason or another do not fit into the classification already given

Mephenesin

Mephenesin (atensin, lisephen, myanesin, relaxar) is β -dihydroxy- γ -(2 methyl phenoxy) propane and consists of colourless crystals with a melting point of 70°C . It is put up as a 10 per cent solution (1 g in 10 ml) which is miscible with thiopentone



Strychnine and picrotoxin antagonize mephenesin to some extent and it was at first thought to act as a depressant on the spinal cord⁵⁶. As however it relieves pain of thalamic origin it seems more probable that its effect is on the basal ganglia⁵⁴. Mephenesin has been observed to check epileptic fits in 30 seconds so that it almost certainly has a central rather than a peripheral effect⁵⁵ (cf curare's time of 90 secs)

An intravenous injection of from 5 to 10 ml in the average fit adult usually produces muscular relaxation with little intercostal or diaphragmatic impairment⁵⁶. Mephenesin has however lost popularity owing to various complications which may occur such as thrombophlebitis⁵⁷ haemolysis followed by haemoglobinuria⁵⁸ and renal failure⁵⁹. It may be that the hypertonicity of the solution or the composition of the solvent may play a part in these sequelae but however this may be the use of the drug is now practically confined to oral administration (as an elixir) or an intravenous drip infusion of about 4 g per litre for spastic states such as tetanus⁶⁰

Dihydro- β -erythroidine

This substance has been extracted from the sub tropical tree *Flame of the Forest*⁶¹ and has been used for some time in the treatment of spastic conditions⁶¹ and for preventing fractures in leptazol convulsive therapy⁶

It appears to act like curare on the myoneural junction but differs in that it is effective when taken by mouth and that it causes a *fall* in blood pressure

In a series of 215 administrations dihydro β erythroidine gave good muscular relaxation for operations with an average total intravenous dose of 240 mg⁶³

Prestonal (Geigy 25178)

This was selected by R. Domenjoz from a number of compounds prepared by Girod and Haefliger in the Geigy Laboratories and has been clinically tested by Rudolf Frey of Heidelberg. Chemically it is N, N, N, N-tetramethyl N, N bis (carbopropoxymethyl) 3, 14 dioxahexadecane 1, 16 diammonium bromide

It appears that the drug should be classed as a neuromuscular blocking agent of the mixed type as it is not reversed by either neostigmine or tensilon, nor on the other hand does it cause muscular twitches and after pain like the suxamethonium halides. It is a powerful inhibitor of the cholinesterases. It requires two minutes to reach its maximal effects after intravenous injection which should be at a rather slow rate to prevent flushing of the face and tachycardia during injection. Its effects last from four to seven minutes⁷⁸

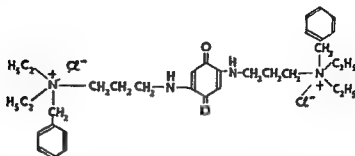
Prestonal has proved useful for obtaining muscular relaxation for short periods for example before endotracheal intubation, for bronchoscopy and for the closure of the peritoneum. A dose of 2 mg/kg will give complete relaxation for approximately five minutes. It appears to have a pronounced relaxant effect on the muscles of the pharynx without depressing spontaneous respiration.

It is put up in 10 ml ampoules of 1 per cent solution, the pH of which is 3.2. In this form it is stable. It does not mix with barbiturates.

Benzoquinonium Chloride

This compound also known as mytolon and WIN 2747 is 2,5 bis-(3 diethylamino propylamino) benzoquinone bis benzyl chloride was synthesized in America⁶⁴ and some clinical work

suggested that it might be useful as a muscle relaxant in man⁴⁵
The solution is coloured red



Two investigations in animals and man in Great Britain have shown that benzoquinonium chloride is undoubtedly an effective relaxant acting for a rather shorter time than curare but that it has various unpleasant side-effects chiefly of a vago mimetic type such as salivation colic, bradycardia and extra systoles⁴⁶ Occasional bronchospasm occurs probably due to histamine release¹ The drug was at first thought to act both as a competitive and as a depolarizing blocker but recent work suggests that the latter effect is slight

Confusion may be caused in the reader's mind by the large number of relaxants described In the writers opinion all reasonable needs can be met by using tubocurarine for prolonged relaxation (usually in divided doses), gallamine for shorter cases and suxa methonium for procedures lasting for a few minutes only

ANTIDOTES

Fairly efficient antidotes exist which reverse the effects of the competitive blockers Most of these belong to the anticholinesterases The same cannot however, be said for depolarizing blockers but since most of these are short acting antidotes are rarely necessary

Neostigmine methylsulphate (prostigmine) has the structural formula —



and appears to act not only by its anticholinesterase effect of increasing the amount of acetylcholine reaching the end plate but also by increasing the responsiveness of the end plate to the small

amount of this substance which can pass the barrier created by the blocker⁶⁷ Unfortunately neostigmine has unwanted parasympathomimetic (muscarinic) side-effects such as bradycardia salivation, pulmonary oedema and diarrhoea These can be largely avoided by giving two minutes previously a large intravenous dose (about gr $\frac{1}{80}$) of atropine The drugs should preferably not be given simultaneously from the same syringe as, at any rate in theory, ventricular fibrillation might occur⁶⁸ Some workers, however, discount this danger⁷¹ The pulse rate should be watched for at least 10 minutes after neostigmine has been given and if it slows excessively, a further dose of atropine should be injected to guard against possible cardiac standstill from inhibition⁶⁸ Neostigmine usually produces quite a dramatic effect at the time but patients may later relapse into a state of inefficient respiration⁷⁰ For this and other reasons it is better practice to ensure that relaxants have lost their effect before the patient is returned to bed than to give an antidote If this is necessary, however neostigmine, e.g. 2.5 mg should be well diluted and given intravenously in divided doses after the preliminary injection of atropine In addition to its anticholinesterase activity it is thought that neostigmine may have a C 10-like effect on the end plate and that a large dose may cause a neuromuscular block

Pyridostigmine (mestinon) is the dimethylcarbamate ester of N methyl pyridium bromide and is an analogue of neostigmine with about half its potency On investigation it is said to have a less reliable effect against competitive blockers than the better-



known drug⁷¹ The nicotinic effect of pyridostigmine on voluntary muscle is more prolonged and its muscarinic effect on viscera is weaker than in the case with neostigmine

Edrophonium (tensilon) is 3 hydroxyphenyldimethylethyl ammonium chloride and is put up in a solution of 10 mg per ml In animals the drug appears to have a complete antagonistic action to the competitive blockers with negligible side effects if atropine is given beforehand In man however, edrophonium is more uncertain and evanescent than neostigmine but it still has some vogue in America⁷² As a considerable part of the pharmacological action of edrophonium is to increase the sensitivity of the muscle end plate to

acetylcholine it does have some slight effect in reversing the paralysis caused by depolarizing blockers. The muscarinic effects are not as marked as with neostigmine.

Other Antidotes

True antidotes to depolarizing blockers are known and have a similar chemical constitution, e.g. both hexa- and pentamethonium reverse the action of decamethonium but unfortunately they are not practicable because of various other effects such as hypotension. Again a compound known as 51-212 antagonizes both competitive and depolarizing blocks but in this case its use in man is debarred owing to its depressor effects.⁴⁷

MORTALITY

A short time ago some concern was felt by the publication of a paper in America concerned with the anæsthetic mortality of 599,548 operations covering a 4-year period in 10 hospitals.⁴⁸ It appeared from the figures given that in 266 deaths where no muscle relaxants had been given the mortality rate was 1 in 2100, while in 118 deaths when a relaxant had been included in the technique the rate was 1 in 370 i.e. nearly six times as high. Furthermore, 74 of these relaxant deaths were attributed to respiratory failure (hypoxia). It is difficult to avoid the conclusion that the prevention of hypoxia and hypercarbia is perhaps not regarded as of the paramount importance it is on this side of the Atlantic. Whatever the reason of these alarming statistics may be, the opinion of responsible anæsthetists in Great Britain is that the introduction of specific muscle relaxants has proved one of the greatest advances of the past twenty years and if properly used the risk of the anæsthetic technique is not increased thereby.

References

1. PAYTON W. M. 1953 *Anæsthesia* July 151
2. RALEIGH, SIR WALTER *Hakluyt's Voyages III* 649
3. BERNARD C. 1865 *Bull. gen. de Thérap.* Paris 69 23
4. WEST R. 1938 *Lancet* Feb 19 432
5. COLE L. 1934 and 1935 *Lancet*
6. KING H. 1935 *Nature* London, 135 469
7. PALMER H. 1948 Aug. personal communication
8. GRIFFITH H. R. and JOHNSON G. E. 1942 *Anesthesiology* 3 418 (July)
9. GRAY T. C. and HALTON J. 1946 *Proc. R. Soc. Med. (An. Sec.)* March 1
10. BULLER A. J. and YOUNG I. M. 1949 *J. Physiol.* 109 412
11. RANSOMOFF N. S. 1947 *Bull. N.Y. Acad. Med.* 23 661
12. KELLGREN J. H. *et al.* 1946 *Brit. med. J.* Dec 14 898

- 13 WINTERBOTTOM E H 1950 *Brit med J* Jan 28 247
- 14 GRAHAM J D P and HEATHCOTE, R 1950 *Brit J Anaesth* Jan 17
- 15 { BAILEY P J 1949 *Anaesthesia* April 52
PATON W D M and ZAMIS E J 1950 *Lancet* Nov 18 568
- 16 Annotation 1953 *Brit med J* April 18 875
- 17 WEST R 1949 *Proc R Soc Med (Sec Exp Med and Therap)* March 8
- 18 GRÖSS E G and CULLEN S C 1945 *Anesthesiology*, May 231
- 19 PRESCOTT F *et al* 1946 *Lancet* July 20 80
- 20 OSTLER G 1947 *Brit med J* April 5 448
- 21 GROB *et al* 1947 *Bull Johns Hopk Hosp* 80 299
- 22 JOHNSTONE, M 1955 *Anaesthesia* April 122
- 23 GRAY T C and GREGORY R A 1948 *Anaesthesia* Jan
- 24 DUNDEE, J W, and GRAY T C 1953 *Lancet* July 4 16
- 25 WILSON A *et al* 1952 *J clin Invest* 31 815
- 26 Memo Anaesthetics Cte M R C, and R S M, 1947
- 27 LEOW E. G 1953 *Arch Surg* 66 312
- 28 GRAY T C 1948 *Proc R Soc Med (An Sec)* April 2
- 29 MORTON H J V and WYLIE W D 1951 *Anaesthesia* Oct 190
- 30 KING H 1935 *J chem Soc* 1381
- 31 COLLIER H O J 1950 *Lancet* June 3 1293
- 32 WILSON H B *et al* 1950 *Lancet* June 3 1296
- 33 MOGEY G A and TREVAN J 1950 *Brit med J* July 22 216
- 34 { BODMAN R J *et al* 1952 *Lancet* Sept 13 517
TAYLOR E P and COLLIER H O J 1951 *Nature* 167 692
- 35 BOVEY D *et al* 1947 *C R Acad Sci Paris* 223 597
- 36 MUSHIN W W *et al* 1949 *Lancet* 726
- 37 DOUGHTY A C and WYLIE W D 1951 *Proc R Soc Med (An Sec)* Jan 5
- 38 { PATON W D M and ZAMIS E J 1948 *Nature* 162 810
ORGANE G 1949 *Lancet* May 7 773
- 39 CHURCHILL DAVIDSON H C 1951 *Proc R Soc Med (An Sec)* Dec 7
- 40 GUERRIER S M and MASON J C 1952 *Brit med J* June 21 1329
- 41 CHURCHILL DAVIDSON H C 1954 *Brit med J* Jan 9 79
- 42 TEWTC G I 1953 *Lancet* July 18 110
- 43 ADAMSON D C and KINSMAN F M 1952 *Anaesthesia* July 166
- 44 { GOULD R B
HURLEY M J and MONRO A B } *Brit med J* 1952 1
HEWER C L } Correspondence
HARPER J K
LOVE, S H S 1952 *Anaesthesia* April 113
- 45 EVANS F T *et al* 1952, *Lancet* June 21 1229
- 46 HODGES R J H and HARKNESS J 1954 *Brit med J* July 3 18
- 47 FOLDES F F *et al* 1953 *Science* 117 383
- 48 BOURNE J G *et al* 1952 *Lancet* June 21 1225
- 49 HERINGTON G and JAMES E 1953 *Brit med J* Aug 8 317
- 50 WHITTAKER and WIESUNDERA 1952 *Biochem J* 52 475
- 51 { WOOLMER H and CATES J E 1952 *Lancet* Oct 25 803
FORRESTER A C 1954 *Brit med J* Aug 7 342
- 52 EDRIIDGE A *Proc R Soc Med (Jt Mig An and Psych Secs)* 1952 June 10
- 53 { BERGER F M and BRADLEY W 1946 *Brit J Pharmacol & Chem* Dec 265
BERGER F M and BRADLEY W 1947 *Lancet* Jan 18 97
- 54 STEPHEN C R and CHANDY J 1947 *Canad med Ass J* 59 463
- 55 HUNTER, A R and WATERFALL J M 1948 *Lancet* Mar 6 366
- 56 MALLINSON F B 1947 *Lancet* Jan 18 98
- 57 GRIFFITH H R., and CULLEN W C 1948 *Anesth & Anal* July-Aug

58. PUGH, J. L., and ENDERBY G. E. H., 1947 *Lancet* Sept. 13 387
- 59 { HEWER, T. F., and WOOLMER, R. F., 1947 *Lancet* Dec. 20 909
- 60 { GOODIER, T. E. W., and GOODHART C. E. 1949 *Lancet* Jan 29 183
- 61 DOCHERTY D. F., 1955 *Lancet* Feb 26 437
- 62 BURMAN M. S., 1939 *Arch Surg & Psych.*, Feb., 307
- 63 ROSEN S. R., 1940 *Psychiat Quart.*, July 477
- 64 DRIPPS, R. D., and SERGENT W. F., 1947 *Anesthesiology* May 241
- 65 { ARROWOOD J. C., 1951 *Anesthesiology* 12 753
- 66 { FOLDES, F. F., 1951 *Anes & Y Acad Sci.*, 54 503
- 67 HOPPE, J. O., 1950 *J Pharmacol.*, 100 333
- 68 { DUNDEE, J. W., et al., 1952, *Anaesthesia* July 134
- 69 { HUNTER, A. R., 1952, *Anaesthesia* July 145
- 70 HUNTER, A. R., 1955 *Brit med. J.*, July 9 128
- 71 JOHNSTONE, M., 1951 *Brit Heart J.*, 13 47
- 72 HUNTER, A. R., 1953 *Brit med. J.*, March 21 640
- 73 SPIERS, R. B., 1951 *Proc Aust Soc Anzsth (N.S.W. Sect.)* Aug. 9
- 74 BROWN A. K., 1954 *Anaesthesia* April, 92
- 75 HUNTER, A. R., 1952, *Brit J Anzsth.*, July 175
- 76 BEECHER, H. K., and TODD D. P., 1954 *Ann. Surg.*, July 2.
- 77 RIKER, W. E., and WESIDE, W. C., 1950 *J Pharmacol.*, 100 454
- 78 O MULLANE, E. J., 1954 *Lancet* June 12, 1209
- 79 CHURCHILL DAVIDSON H. C., 1955 *Proc Roy Soc Med.*, 48 621
- 80 HUNTER A. R., 1953 *Brit med. J.*, March 21 640
- 81 JOLLY C., 1957 *Anaesthesia* Jan. 3

CHAPTER 8

LOCAL ANALGESIA

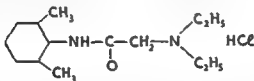
Toxic Effects of Local Analgesic Drugs—Some Newer Local Analgesic Drugs—Hyaluronidase—Drugs Used for Vasoconstriction—Some Newer Pressor Drugs

Untoward Reactions to Local Analgesia Drugs

THESE were formerly divided into those affecting the nervous system which resulted in death from convulsions and from respiratory failure the result of muscular inco-ordination, and those causing acute circulatory collapse. Recently a more detailed classification has been made into toxic manifestations in normal individuals, abnormal responses and effects not directly due to the local analgesic drug itself¹. Included in the first group are central nervous system effects such as stimulation of the cortex and depression of the respiratory and vasomotor mechanisms and cardiovascular effects such as direct action on the heart and action on the vascular bed. Abnormal responses include allergy, hypersensitivity and idiosyncrasy. The associated reactions are likely to be due to fear and anxiety or to the effects of the pressor amine used to promote vasoconstriction. Any of these may occur singly or they may be combined.

Some Newer Local Analgesic Drugs

Lignocaine (xylocaine, xylotox, lidocaine, lastracaine) This is diethylamino 2,6-dimethylacetanilide hydrochloride

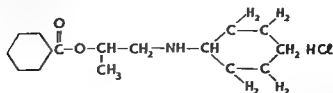


It was synthesized in 1943 by Lofgren and Lundquist. It differs in its chemical structure from cocaine and procaine being a derivative of acetanilide. It is the best all purpose local analgesic drug in use at the present time for infiltration, nerve block and surface analgesia. Its toxicity is greater than that of procaine in the stronger solutions but it is more potent so that its therapeutic index is higher. It is extremely stable, is not decomposed by alkalis or acids, will withstand repeated autoclaving, is a rapidly spreading and diffusing drug.

and produces most effective analgesia. It can be successfully used for all types of pain relief: spinal, extradural, nerve block, infiltration and topical. It is without effect on the calibre of small blood vessels, being neither a vasodilator nor a vasoconstrictor. It can be used in those patients who react badly to procaine and its congeners. The drug is probably excreted by the kidneys after hydrolysis by a plasma esterase, but this occurs less rapidly than is the case with procaine.

For infiltration 0.5 per cent solution is adequate, with a maximum dose of 100 ml (500 mg). In extensive cases, half this concentration has been used with success. The strength for application to the cornea is 2 per cent, and that for topical analgesia of the mucous membrane of the upper air passages is 2 per cent to 4 per cent. In dentistry 2 per cent solution with 1:50,000 or 1:80,000 adrenaline is used. Lignocaine, and other local analgesics put up with adrenaline, must be made acid (pH 4) in order to remain stable, and this acid reaction may have a harmful effect on the metal of syringes so that the solution should not be stored in a syringe or metal container, but should be used immediately and the syringe (or container) afterwards carefully washed. Xylodase is 5 per cent lignocaine in a water-soluble base of carbowax to which is added hyaluronidase to promote rapid action and depth of penetration. It is a useful lubricant for endotracheal tubes and serves to deaden the pain of dental injections of local analgesic drugs if rubbed on to the gum. Xylocaine Viscous contains 2 per cent of the analgesic in a palatable base which produces when swallowed a degree of analgesia of the mouth, pharynx and œsophagus prior to laryngoscopy, œsophagoscopy and gastroscopy when these examinations are to be made without general anaesthesia. Its high viscosity and low surface tension enable it to have a prolonged and useful effect on mucosal surfaces, including that of the intestine in patients who complain of dumping syndrome after gastrectomy. The dose varies from a teaspoonful to a table-spoonful. Xylocaine Gel contains 2 per cent of the drug and is a safe and efficient agent for the production of analgesia in urethral instrumentation.

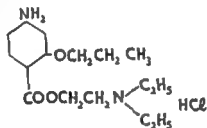
Hexylcaine (cyclaine)



This is 1-cyclohexylamino 2-propylbenzoate hydrochloride and was synthesized by Cope and Hancock³ in 1944. The 1 per cent

solution can be autoclaved and has a low toxicity relative to procaine and surface effects similar to cocaine. It combines the long duration of action of amethocaine with moderate chemical stability. For infiltration 1 per cent solution can be used while 5 per cent solution gives good topical analgesia. For subarachnoid block, 1.5 per cent solution made hyperbaric with 10 per cent glucose has been successfully used. The pH of a 1 per cent solution is 3.9.⁴ Its use as a topical analgesic applied to the larynx is said to reduce the incidence of arrhythmia associated with laryngoscopy and intubation,⁵ cocaine and other topical analgesics make these arrhythmias worse. Other investigators have found the agent causes irritation and some residual soreness when used for infiltration,⁶ but on the whole this new drug has been found to be a useful addition to the range of local analgesics.⁷

Ravocaine (pravocaine)



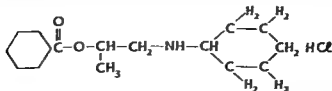
This is procaine with two propoxy groups and is a white crystalline solid melting at 148° C, readily soluble in water with analgesic activity half way between that of procaine and cocaine. The pH of a 1 per cent solution is 5.5. Its topical activity is greater than that of cocaine, while its toxicity is comparable to that of amethocaine. For infiltration 0.1 per cent solution and for nerve block 0.5 per cent solution is suitable. For subarachnoid block 0.3 per cent with 10 per cent glucose has been used,⁴ and good analgesia has been reported.⁸ Dental surgeons have had good results following the injection of 0.4 per cent solution.⁹

2-Chloroprocaine This is a white amorphous powder with molecular weight 307.21 and a melting point of 170° C. It was described by Foldes in 1952.¹⁰ The salt dissolves fairly well in water and the 1 per cent solution has a pH of 4.9. It is moderately stable at room temperature and has a high therapeutic index. It is rapidly hydrolysed by the pseudocholinesterase of plasma, the rate being four times that of procaine. It has been successfully employed in spinal analgesia in which the successful results are higher and the onset more rapid than when procaine is used. It is reported to have a greater penetrating power than procaine. It is sold commercially as nesacaine.

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Hexylcaine (cyclaine)



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test of time and experience, as serious neurological sequelæ have been reported after its use. One patient developed transverse myelitis following the injection of a small volume into the intercostal nerve 3 in from the midline. Other workers have seen cases of toxic neuritis.¹¹ Experimental work has shown that the propylene glycol is largely responsible for the destruction of nerve tissue, and incidentally for the analgesic action.^{12, 13} The drug must therefore be used, if at all, with the very greatest care.

The Use of Hyaluronidase with Local Analgesics

Hyaluronidase (hyalase, rondase, hydase, wydase, diffusin) is a mucolytic enzyme which hydrolyses or depolymerizes hyaluronic acid. It increases the area of the distribution of drugs in the tissue spaces and therefore aids their absorption and so is useful for hypodermoclysis in infants and children. In local analgesia it facilitates penetration of solution into nerve fibres and causes a speedier onset and a larger area of analgesia with the use of a smaller volume of solution. With these good points are some bad ones: it results in a shorter duration of analgesia and perhaps an increased toxicity of the drug used. As a mucolytic agent it reduces the time of onset of analgesia if incorporated in an analgesic cream for topical use, as in xylodase. Vasoconstrictors increase the spreading effect by delaying the absorption of the enzyme. The usual dose employed is one turbidity reducing unit (TRU) for each 1 ml of analgesic solution. It finds its greatest use in extensive infiltration analgesia and in internal pudendal block and blocks for fractures such as Colles fracture. For lumbar extradural block it should be avoided. Successful nerve blocks are better obtained by a knowledge of anatomy than by the indiscriminate use of this enzyme. An international hyaluronidase standard has now been defined by the World Health Organization at Geneva (1955) so for example 3 mg of rondase equals 1000 international units. One thousand Benger units roughly equal 450 turbidity reducing units or 200 viscosity reducing units. For hypodermoclysis, 1000 units of hyalase are sufficient for the absorption of 500-1000 ml of fluid.

Drugs Used for Vasoconstriction in Local Analgesia

In addition to adrenaline and cobefrin, phenylephrine (ne-synephrine) has been successfully used to prevent the too rapid absorption of local analgesic solutions. It differs from adrenaline chemically in that it has one instead of two hydroxyl groups on the benzene ring—in the meta position. Synephrine, an older drug had



FIG 55 Extreme dissociation in the nerve fibres 11 days after intraneural injection of Efoaine (Mannheimer *et al JAMA*, 1954)

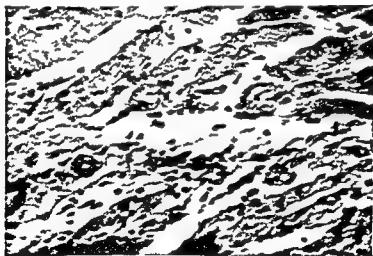


FIG 56 Degeneration and necrosis of the muscle 48 hours after injection of Efoaine (Mannheimer *et al JAMA* 1954)

Efoaine This contains 1 per cent procaine base □ 25 per cent procaine hydrochloride 5 per cent butyl para aminobenzoate dissolved in polyethylene glycol 300 and 2 per cent propylene glycol (78 per cent in water) It was introduced as a long acting local analgesic solution free from toxicity but has not withstood the

pressure following bromethol administration. When added (0.25–0.5 ml) to a solution of spinal analgesic, it increases the duration of pain relief, as does adrenaline (0.25 ml of 1:1000 solution).

Methoxamine (vasoxine, vasoxyl, vasylox) This chemically is β hydroxy β (2, 5 dimethoxyphenyl) isopropylamine and is a very promising new drug. A vasopressor by virtue of its action on vessels, it influences neither the heart rate and output nor the cerebral cortex. It can be used on patients under the influence of cyclopropane without the risk of precipitating an attack of ventricular fibrillation. Bradycardia from aorticocarotid sinus reflexes, accompanies the rise in pressure which comes on very soon after intravenous injection of 2–4 mg which can be repeated. The intramuscular dose is 5–10 mg, the onset occurring in fifteen minutes, and the duration of action, one hour. It has no effect on the bronchial musculature, but causes pilomotor stimulation. It is put up in 1 per cent solution.

Mephenteramine sulphate (wyamine, mephine) Chemically this is N methylphenyl tertiary butylamine. It is inactive optically. The drug has been studied by Brotman¹⁵ who found it to be a vasopressor acting on the vessels. In the dog it has been shown to exert a positive inotropic and chronotropic effect.¹⁶ It is a long acting drug both after intravenous and intramuscular injection. It has no anorexic or cerebral stimulating effects. The intravenous dose is 15 mg given slowly and repeated if necessary. It is put up in 1.5 per cent solution and also as an inhalant to reduce nasal congestion and hyperæmia. It can be given as an intravenous drip, and has achieved success in the immediate treatment of some cases of hypotension associated with coronary occlusion. It is reported that mephenteramine protects against ventricular fibrillation—though not against tachycardia, hypertension or extrasystoles—in animals under cyclopropane anaesthesia who receive an otherwise fatal dose of adrenaline.

These drugs are all useful to reverse the hypotension produced by spinal or extradural analgesia and to combat certain cases of shock when given together with blood plasma or plasma volume expanders.

Isopropylnoradrenaline (neoepine, isoprenaline, isoproterenol, isopropylnorarterenol, aleudrine, isuprel, norisodrine) This drug although a congener of adrenaline is not used as a vasoconstrictor. It has its effects largely on the bronchi and so is useful in the relief of bronchospasm. On intravenous injection it causes vasodilatation with consequent hypotension, an effect lasting from five to fifteen minutes. It reduces the peripheral resistance in the renal and splanchnic regions while its cardiac effects are similar to those of

the hydroxyl group in the para position. It produces good vasoconstriction and has no effects on either the cerebral cortex or the heart and thus causes neither excitement nor tachycardia. It is put up in 1 per cent solution and is used in a final strength of 1 : 2000 or 1 : 2500.

Some Newer Pressor Drugs

TABLE SHOWING CHEMICAL RELATIONSHIP OF SOME VASOCONSTRICTORS

					Adrenaline
OH	OH	OH	H ₂	H	Noradrenaline
OH	OH	OH	H ₂	CH(CH ₃) ₂	Isopropyl Noradrenaline
H	OH	OH	H ₂	CH ₂	Phenylephrine
H	H	OH	HCH ₃	CH ₂	Ephedrine
H	H	H	HCH ₃	CH ₂	Methyl Amphetamine
H	H	H	(CH ₃) ₂	CH ₂	Mephenteramine

The table gives the chemical relationships of some commonly used vasoconstrictor drugs.

Phenylephrine (neosynephrine, isophryn, neophryn, metasymptol). This is an optically active pressor amine differing only slightly from adrenaline chemically (see above) and pharmacologically. Its chief effect is on the circulatory system and the intravenous injection of 0.8 mg intravenously or of 5 mg intramuscularly causes a rise first in diastolic then in systolic blood pressure which lasts for twenty to sixty minutes. With the hypertension is associated bradycardia which is probably a reflex effect from the baroreceptors in the aortic carotid sinuses but may be a direct cholinergic effect on the S-A node.¹⁴ It does not cause many cardiac irregularities with cyclopropane and can be given in an intravenous drip. It is issued in 1 per cent sterile solution each ml containing 10 mg. It produces no tachyphylaxis and does not stimulate the central cortex. It has been used to combat paroxysmal auricular or nodal tachycardia in normotensive patients and is accompanied by a minimum of side actions. It can be used as an inhalation in asthma and serves to raise the blood

ml of solution are added to 500 ml of saline, dextrose solution or plasma expander, the resulting strength will be 4 μ g per litre. The solution is employed as a continuous intravenous drip, the rate being regulated according to blood pressure readings.

It has been successfully used in the treatment of peripheral circulatory failure, together with blood or plasma expanders in surgical shock, and in circulatory failure consequent on coronary occlusion after pulmonary embolism removal of chromaffin cell tumours, and after certain cases of bilateral adrenalectomy. It can raise the lowered blood pressure of patients in whom other hypertensive drugs such as methoxamine, phenylephrine and methylamphetamine have failed. It decreases the renal blood flow, unlike methedrine which increases it¹ and also reduces the blood flow through the liver and brain.

Antagonists to toxic doses of adrenaline and noradrenaline (and 0.5 mg of the former injected subcutaneously has proved fatal) include the following group of drugs. The ergot alkaloids, the benzyl chloroethylamines such as dibenamine, the imidazolines e.g. tolazoline (priscof), the phentolamine group e.g. regitine and the benzodioxanes e.g. piperoxan. Of these the most useful in clinical work are likely to be piperoxan 15 mg intravenously or phentolamine, 5–10 mg. Cholinergic compounds—the pharmacological antidotes, may also be helpful, e.g. carbachol or mecholyl.

References

- 1 SADOVE M S *et al* 1952 *J Amer med Ass* 148 17
- 2 LÖFEGREN N 1946 *Ark Kemi Min Geol* 22A No 18
- 3 HANCOCK E M *et al* 1944 *J Amer chem Society* 66 1747
- 4 CRAWFORD O B 1953 *Anesthesiology* 14 278 (May)
- 5 ARCURI R A *et al* 1953 *Anesthesiology* 14 46 (Jan)
- 6 ANDERSON E 1952 *Anesthesiology* 13 429 (July)
- 7 ORKIN L R 1952 *Anesthesiology* 13 465 (Sept)
- 8 SADOVE M S *et al* 1953 *Amer J Proctol* (Dec)
- 9 DOBBS E C *et al* 1954 *J Amer dent Ass* 48 409 (April)
- 10 FOLDES F F *et al* 1952 *Anesthesiology* 13 289 (May)
- 11 SHAPIRO S M and NORMAN D D 1953 *J Amer med Ass* 152 608
- 12 MAYKUT M O and RYAN E L 1953 *Canad med Ass J* 69 419
- 13 MANNHEIMER W *et al* 1954 *J Amer med Ass* 154 29
- 14 KEYS A and VIOLANTE A 1942 *J clin Invest* 21 1
- 15 BROTMAN B L *et al* 1952 *Amer Heart J* 44 496
- 16 GOLDBERG L I *et al* 1953 *J Pharmacol* 108 177
- 17 LANDS A M and HOWARD W J 1952 *J Pharmacol* 106 65
- 18 GOLDENBERG M *et al* 1949 *J Amer med Ass* 140 771
- 19 v EULER U S 1955 *Lancet* 2 151
- 20 GAZES P C *et al* 1953 *Circulation* 8 883
- 21 DE WARDENER H E 1955 *Anaesthesia* 10 18 (Jan)
- 22 LYNCH P R *et al* 1955 *Anesthesiology* 16 632 (July)
- 23 LITTLE, T R and MCKENDRICK, C S 1957 *Lancet* 2 825 (Oct 26)

adrenaline that is it has a positive inotropic and chronotropic effect¹⁷ Its chief clinical use is to dilate the bronchi in bronchial asthma, while it also inhibits peristalsis and intestinal tone It has a mildly stimulating effect on the cortex It is probably oxidized by an enzyme similar to amine oxidase It is prepared in 5 and 10 mg tablets for sublingual use and in 0.5 and 1 per cent solution for inhalation as an aerosol While cardiovascular side effects are not infrequent after its sublingual administration these are rare after inhalation

Noradrenaline (l-arterenol, levophed) This new drug, like adrenaline, a catechol amine, was synthesized in 1904 by Stolz. It is probably the chemical mediator of sympathetic nerve impulses Chemically it is similar to adrenaline, only the methyl radical attached to the nitrogen atom in the former drug has disappeared in noradrenaline, hence the term nor (nitrogen ohne radikal) It was first used clinically in 1949 by Goldenberg and his colleagues¹⁸ who employed it in states of hypotension and shock It is a regular constituent of the post ganglionic sympathetic nerves and is found in all organs supplied by such nerves It is also present in the adrenal medulla It raises both the systolic and diastolic blood pressure dilates the coronary arteries¹⁹ and this effect, together with the increase in aortic pressure which it produces enables it to act as a potent heart stimulant²⁰ While adrenaline acts as the fight flight and fright hormone and is concerned with alterations in metabolism noradrenaline is the pressor hormone contracting not only the vessels in the skin and subcutaneous tissues but also those in muscle but causes a bradycardia reflexly from the aortic and carotid sinuses It directly increases the contractile force of the myocardium but may make it more irritable²¹ It is oxidized by amine oxidase rather more quickly than is adrenaline and so is not very satisfactory when added to solutions of local analgesic and when injected with saline to produce localized ischaemia Extravenous leakage of a drip infusion into small peripheral veins has been known to cause tissue necrosis so it should be run into the proximal and larger veins or carried up to them through a plastic cannula Although less likely to initiate ventricular arrhythmias than is adrenaline its use in conjunction with cyclopropane is probably unwise Vessels gradually lose their responsiveness to physiologically released and artificially injected noradrenaline during prolonged low blood pressure states so active resuscitation with this drug must be started early¹⁹

Noradrenaline is put up commercially as the bitartrate in ampoules of 1 : 1000 solution each ml containing 1 mg of the drug If two

As the method is not very extensively employed, recent advances are few, and at present this is not one of the fastest growing points in our specialty.

The effects of spinal analgesia on the circulation have been carefully studied and it has been shown that there is reduction in stroke volume and cardiac output, and reduction in right auricular, pulmonary arterial and brachial arterial pressures, a decrease in peripheral resistance, rate of oxygen consumption and heart rate, a fall in the oxygen content in the pulmonary artery and the brachial artery. There is no decrease in the arterial blood oxygen saturation and the arteriovenous oxygen difference is increased. All these changes are more marked in high than in low block and are due to vasomotor paralysis.

The estimation of the specific gravity of cerebrospinal fluid is more complicated than would appear. Water chloride, sodium and carbon dioxide affect density to the fourth or less place of decimals whereas albumen and globulin affect it only to the fifth place. If a solution to be injected into the theca has a density at 37° C greater than 1.0022 g per ml it can be regarded as hyperbaric and if it is less than 0.9998 g per ml it can be treated as hypobaric. At 37° C human cerebrospinal fluid is said to have a density of 1.0010 ± 0.0003 g per ml or 1.0040 ± 0.0004 g per ml at 25° C.³ Cerebrospinal fluid contains small quantities of cholinesterase but this probably does not cause appreciable hydrolysis of agents such as procaine, lignocaine and amethocaine and so does not make analgesia less intense when these agents are used.

HIGH (TOTAL) SPINAL BLOCK

High spinal block still retains its popularity in some centres as a ready means of ensuring relaxation, analgesia and ischaemia. While procaine was used originally by Griffiths and Gillies,⁴ hyperbaric nupercaine or other drugs can be used. With this technique scrupulous attention must be paid to the ventilation of the patient, an endotracheal tube being used if necessary, and the condition of the circulation must be constantly under review. Intravenous injection of a pressor drug may be needed⁵ if there is alteration of the depth or rhythm of respiration, prolongation of capillary filling time, cyanosis in the presence of a good airway and oxygen tension in the inspired atmosphere, a sudden fall in the blood pressure after its initial stabilization, and gross alteration in the rate or rhythm of the pulse. The technique is probably contra-indicated in patients with coronary disease, severe arteriosclerosis, aortitis and aortic

CHAPTER 9

THE PRESENT POSITION OF SUBARACHNOID AND EXTRADURAL ANALGESIA

IN the United Kingdom there is at the time of writing a cloud over spinal analgesia. This is not because it is an unsatisfactory method of pain relief, while the fact that it is often accompanied by hypotension is now regarded as one of its advantages when formerly this was looked on as an undesirable event. It is the neurological complications of which anaesthetists are afraid and this fear is partly due to the increase in litigation since 1948 and to some recent decisions in the High Court e.g. the Woolley and Roe case.¹ Uneasiness is made worse by the fact that the cause of serious neurological complications is not fully understood. While neurologists tend to blame susceptibility of the nervous tissue of certain patients to the drugs injected, surgeons think first of infection as the cause and anaesthetists—or some of them—have suggested that minute flaws in the glass ampoules containing the local analgesic solution have allowed strong antiseptic in which storage has taken place to seep in and be injected intrathecally. Sir Francis Walsh states categorically² that the cause is the local analgesic drug and quite rightly points out that the immersion of ampoules in toxic solutions for purposes of sterilization is by no means a constant factor in the recorded cases of post spinal neurological complications. There may well be more than one cause for this most grave complication and research into this matter is urgently necessary. Added to this the missionary work of Massey Dawkins, Bromage and others in popularizing lumbar extradural analgesia has focused attention away from spinal block. They have shown that their method gives most of the advantages of spinal analgesia without producing the occasional untoward result associated with it. Clouds pass however and it is possible that the method will once again enjoy at least some of the warm rays of popularity. The dangers and management of hypotension are now better understood than they were while auto claving of the entire armamentarium should avoid chemical and bacteriological adulteration of the injected solution. The favourable report of Dripps and Vandam (see later) is perhaps the first move in this direction.

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the fifth lumbar vertebra and the sacrum into the subarachnoid space and through it supplementary doses of local analgesic solution are injected. It seems doubtful if orthopedic surgeons would appreciate this new technique being employed on their patients as theoretically posterior protrusion of the disc might result.

The complications of spinal analgesia prevent the method from achieving real popularity and foremost among these is the post spinal headache. Most workers now believe that this is due to leakage of cerebrospinal fluid through the hole in the dura into the extradural space in patients who are not able to secrete sufficient fresh fluid to maintain an adequate fluid buffer for the brain and cranial nerves. It appears that headaches of this type are more than twice as common in females than males.¹⁰ The use of a pencil pointed lumbar puncture needle of small calibre with a lateral opening near the tip which has no cutting edge¹¹ is said to lessen the incidence of headaches¹² and the use of a fine gauge needle also has a beneficial effect on the incidence of this complication.¹³ On the other hand in a controlled series of 610 cases it was not possible to show any difference resulting from the use of a fine needle.¹⁴ Post spinal headaches are said to be reduced in frequency if the needle is inserted into an unflexed back, so that the hole made in the dura when slack is smaller than if the dura is stretched.¹⁵

Paralysis of the sixth nerve may well have a similar cause to post spinal headache and the condition is probably always preceded by headache. For this reason post spinal headache should be treated seriously and the patient must be put to bed and nursed supine without pillows. Fluids should be given in large amounts either by mouth or intravenously until the headache goes. Posterior pituitary extract 1 ml. should be injected intramuscularly three daily for its antidiuretic effect.¹⁶

Serious Neurological Complications These have been reported all too frequently after spinal analgesia but if scrupulous care is taken over cleanliness and sterilization of equipment and if only well established drugs are employed then they should be rare. Chemical meningitis can be provoked by the smallest trace of lysol introduced by a needle,¹⁸ while minute quantities of detergent used for cleaning syringes and needles before autoclaving are believed to be capable of causing adhesive arachnoiditis.¹⁷ All are agreed that a safe method is to autoclave syringes and needles at 115° C which equals 10 lb for 30 minutes but repeated autoclaving of ampoules of drugs may in the case of procaine, amethocaine and cinchocaine, lead to some loss of strength, although no toxic decomposition products

valvular disease, congenital heart disease, severe anæmia, polycythæmia, and in conditions resulting in inadequate respiration, decreased cerebral blood flow and peripheral vascular collapse. To these must be added those conditions in which subarachnoid block of all kinds is thought to be inadvisable.

The indications for spinal block are non-existent in some quarters, and very wide in others. In an able discussion dealing with this matter at the Royal Society of Medicine in 1954⁶ the following operations were mentioned as being reasonably suitable—abdomino-perineal resection of the rectum, prostatectomy and amputation of the leg in certain old, arteriosclerotic or diabetic patients. In operative obstetrics the method is perhaps shown at its best. It is well known that aspiration of gastric contents by women in labour accounts for many deaths each year and if a general anæsthetic is given it is difficult to prevent this in every case. The patient who receives a low spinal such as 0.8 ml. of hyperbaric cinchocaine solution for forceps delivery, remains in full control of her cough reflex throughout the proceeding, even should vomiting occur. A higher block can be successfully used in Cæsarean section (e.g. 1.4 to 1.6 ml. of the same solution) and provided the blood pressure is maintained at a figure above 90 mm. of mercury, good results are the rule. In Thorne's series of 500 cases of operative delivery⁶ the method has given every satisfaction to patient, obstetrician and anæsthetist. In a series of 1236 patients who underwent Cæsarean section in San Francisco de Carle⁷ was impressed with the advantages of the method of analgesia—lack of aspiration of gastric contents, benign effect on the foetus especially if premature and good relaxation and relief from pain. He never gave more than 50 mg. of procaine with 10 mg. of amethocaine and all his patients had from the outset an open vein readily available for injection of fluid oxytocics, pressor drugs, blood or intravenous anæsthetics. He warns against giving ergometrine to a patient who has just received ephedrine because of the very steep rise in blood pressure which may sometimes result from a combination of the two drugs. The patient who is having an operation under spinal analgesia may retch or vomit especially if the blood pressure is allowed to fall too low but the distressing symptoms may sometimes be prevented by intravenous injection of chlorpromazine before the spinal injection is given.⁸ A new method has been described for prolonging the effects of subarachnoid block.⁹ With the abdomen open and the patient in a steep Trendelenburg position a fine needle is inserted directly backwards through the intervertebral fibrocartilage between

It is an interesting fact that neurological sequelæ to surgical operations are not confined to those done under spinal analgesia. A long lasting case of diplopia followed thiopentone curare and cyclopropane²¹ and a case of ascending spinal paralysis which ended fatally came on after an operation performed under thiopentone, gas oxygen ether and gallamine²². Paralysis of the peroneal nerve is not very rare after general anaesthesia while it is not always easy to explain some cases of brachial plexus palsy after general anaesthesia. Additional reports on the safety and satisfaction associated with spinal analgesia come from Scandinavia²³ and the United States.⁴ These must however be set against a further series of 12,000 cases of saddle block analgesia used for delivery in obstetrics.²⁴ Over a three year period in St. Louis, Mo., 6 cases of adhesive arachnoiditis were discovered—an incidence of 0.47 per cent. It would seem that in all these cases the analgesic was quite properly administered the drugs used being 0.25 per cent cinchocaine in 5 per cent dextrose and 1.5 per cent, and 3 per cent piperocaine (metycaine). There was no hospital mortality in these cases and the alarming fact appeared that neurological symptoms did not occur until at least six weeks after discharge from hospital. With such results it is not surprising that saddle block analgesia in obstetrics has now been given up in the hospitals concerned.

A case of paraplegia coming on seven days after spinal analgesia has recently been reported. 20 ml of hypobaric cinchocaine, the pH of which is 4.5 or less, was injected. One cause of the arachnoiditis present in this case might have been the acidity of the solution, or the body's inability to neutralize this acidity.⁴⁷

The therapeutic uses of spinal analgesia are now almost confined to the long term destruction of afferent nerve fibres in patients with incurable pain. While absolute alcohol was formerly the drug most frequently used phenol dissolved in glycerine has been used recently.²⁵ 1 to 1.7 ml of a solution containing one part of phenol in 18–20 parts of glycerine with 0.1 ml of distilled water to each ml of solution is satisfactory. It destroys the small non-medullated C fibres, the larger A proprioceptive fibres being undamaged. The solution is strongly hyperbaric the specific gravity of glycerine being 1.250. After lumbar puncture in the lateral position with the painful side downwards the patient is rotated backwards so that the posterior root ganglia will be bathed in solution. The location of the needle is checked by injection of hyperbaric cinchocaine solution and if this causes localized analgesia of the painful area it is followed by the injection of the glycerine-phenol solution. The needle is left in situ

have been demonstrated. Lignocaine tolerates repeated autoclaving well. When autoclaved at 250° C at 20 lb pressure for thirty minutes on five separate occasions, changes took place in solutions as follows: procaine 200 mg in 10 per cent solution became 196.2 mg, amethocaine 20 mg 1 per cent solution became 19.6 mg, amethocaine crystals 20 mg became 19.45 mg and piperocaine 150 mg in 5 per cent solution became 149.7 mg.¹⁸ There is not much loss of strength here. This severe heat treatment produced some caramelization in glucose solutions, but so far no harmful effects have resulted from their intrathecal injection.¹⁸ A recent observer, stimulated by the Woolley and Roe case, autoclaved batches of ampoules at 10 lb pressure for one to six hours, and later examined samples of each batch for appearance, content of active constituent, pH, toxicity, and analgesic potency.¹⁹ It is concluded from these experiments that lignocaine is a very stable compound whereas cinchocaine undergoes slight, and amethocaine considerable decomposition on prolonged sterilization. Heavy cinchocaine commenced to deteriorate in the fourth hour and its light solution in the second hour. No increase in toxicity was found with either cinchocaine or amethocaine. Repeated or prolonged sterilization of ampoules is thus undesirable, but if each ampoule is placed in a small screw capped glass container before its first autoclaving it can be kept ready for use so that both wastage of ampoules and repeated autoclaving can be avoided. It has been suggested that exposure to formaldehyde vapour is a safe method of sterilization if autoclaving is harmful, but small cracks in the glass may admit this irritant into the ampoule where its vapour might dissolve in the contents with grave results.

A good word for spinal analgesia comes from a most carefully followed up series of over 10 000 cases of block performed in Philadelphia.²⁰ Minor neurological sequelæ of sufficient frequency and severity to suggest strongly a causal relationship to spinal analgesia were recorded. These included backache, pain and numbness in the buttocks, thighs, legs and feet and occasional weakness in the leg muscles. The majority of these minor complications were transient and disappeared completely with the passage of time. A good many of these findings could be related to the lumbar puncture. In the whole series of over 10 000 cases no major complication was found while the minor ones instead of getting worse improved. These investigators were of the opinion that mortality rates were lower after spinal analgesia than after general anaesthesia in comparable patients and operations.

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for one hour so that additional injections can be made if necessary. Should the solution fail to produce the desired result it can be replaced with 1 ml of glycerine containing 0.6 mg of silver nitrate. For pain of pelvic origin the needle should tap the subarachnoid space between the twelfth thoracic and first lumbar vertebra. Pain in the sciatic distribution should be attacked from the second lumbar interspace and sacral pain from the fourth. Pain in the abdomen and in the chest can be dealt with from higher levels. Paralysis of the legs and in rectal cases, incontinence, are occasional complications, but in spite of these the method is considered a useful means of dealing with a grave problem. Spinal analgesia has been used with success in the treatment of the thyroid crisis, the aim being to denervate the suprarenal glands and so remove the driving force from the thyroid.

LUMBAR AND SACRAL EXTRADURAL ANALGESIA

One of the most significant changes in the climate of opinion within recent years has been the decrease in favour of subarachnoid block, but along with this has been a revival of interest in extradural analgesia. Many anaesthetists who formerly used spinal analgesia have now discarded it in favour of extradural block, their reasons being three in number. First and foremost, several cases in the High Court have drawn attention to the small but definite risk of neurological complications after the most carefully given spinal injections. Secondly the increasing use of lignocaine (xylocaine) with its reputation for solid reliability has made the technique so much more certain than was the case when procaine was used. Lastly the slight technical difficulty of making a successful extradural block has acted as a stimulus and has aroused not only interest but enthusiasm in the minds of many anaesthetists in recent years. Lumbar extradural block is very satisfying to perform. Add to this profound analgesia, excellent muscular relaxation, adequate spontaneous breathing and a definite, albeit uncertain degree of hypotension and almost complete absence of sequelae and it is obvious why the method has become popular. In the United Kingdom the writings of Massey Dawkins²⁷ Bromage⁸ and others have stimulated interest and added to our knowledge of the method. The solution of local analgesia exerts its effect in one or all of three situations, on the anterior and posterior nerve roots in the extradural space, on the nerve roots in the paravertebral spaces after they have left behind their dural sheaths and on the nerve roots in the subarachnoid space following diffusion of the solution through the dura and arachnoid.

Methods of Identifying the Extradural Space

In the opinion of the writers, the loss of resistance test, popularized by Dogliotti is the surest means of identifying the extradural space. Loss of resistance to injection of liquid or air is most easily appreciated if a wide bore needle is used, e.g. 18 or 19 H.W.G. and if this has a short bevel and is blunt, then dural puncture becomes less likely. Through a wheal of local analgesic solution the needle is pushed forwards between two easily palpable spinous processes, until it is held firmly in the interspinous ligament. Thereafter advancement is millimetre by millimetre, with frequent pauses to test the resistance to injection. When the blunt short bevelled point enters the extradural space from the dense yellow elastic ligamentum flavum resistance to injection dramatically and suddenly disappears. If in addition rotation of the needle coughing by the patient and aspiration all fail to demonstrate the presence of cerebrospinal fluid it can fairly safely be assumed that the needle has found its mark. A test injection of 5-10 ml. of analgesic solution is an additional safeguard. The distance between the skin and the extradural space averages 3 to 5 cm. The lateral position is satisfactory for most patients; the sitting position for the obese and those with lordosis.

The hanging drop sign of Gutierrez,²⁹ depending as it does on the demonstration of the negative pressure in the extradural space is not sufficiently reliable for routine use. Anyone doubting this should try to elicit it every time he does a subarachnoid block. Odom's indicator³⁰ has its advocates, while several other cleverly designed pieces of apparatus have been invented to help the anaesthetist to know when he is safely home.^{31, 32} The Iklé³³ spring loaded syringe automatically injects some of its contained solution when the needle enters the space. The negative pressure in the extradural space is greater at high thoracic than at low lumbar levels and while in the former case it is probably due to the negative intrathoracic pressure in the latter it is accounted for by the dimpling of the dura by the advancing needle. It is important to choose a palpable and well defined lumbar interspace. Its site high or low is relatively unimportant and can be compensated for by a little more or a little less solution injected. Insertion of the needle in the thoracic region is more critical than it is in the lumbar region and a negative pressure sign (e.g. Odom's indicator) should be used rather than the lack of resistance test while the patient should be sitting up. In this region the dura is closer to the ligamentum flavum than it is lower down and injury to the dura and even to the cord will occur if great

delicacy of touch is not used. Mid thoracic puncture is difficult and is perhaps better avoided. If the dura is perforated, some workers advise trying again in an adjacent interspace³⁴ but others abandon the method or give an ordinary spinal injection.

The commonly used local analgesic drugs can be employed but lignocaine is rapidly establishing itself as a firm favourite for this as for many other purposes. The solution is stable and can be autoclaved repeatedly. Its effects are rapid in onset—about five to ten minutes—and reasonably prolonged in duration—2–2½ hours using the 1.5 per cent solution. It takes a firm ‘bite’ on the nerve roots, causes some somnolence following its absorption into the general circulation and is not very often guilty of causing toxic effects under ordinary clinical conditions. Bromage²⁸ believes in the ability of a 1.2 per cent solution to give a differential block i.e. a sensory analgesia with reflex motor atony but without direct muscular paralysis such as intercostal block. In the writers’ opinion 1.5 per cent solution allows very good respiration to take place, while it is rather more certain and sure in action than the weaker solution. A 2 per cent solution seems unnecessarily strong. When a longer period of analgesia is required because of the expected duration of the operation or because prolonged post operative analgesia is thought to be desirable, amethocaine hydrochloride can be added to the lignocaine solution. A convenient way of doing this is to add half the contents of one 100 mg ampoule of crystals to 50 ml of solution, giving a final strength of 0.1 per cent. Adrenaline is always used with solutions injected into the extradural space, both to reduce toxicity from rapid absorption into the blood stream and to prolong the effect of the analgesic. 0.25 ml can be added to 50 ml of solution giving a final strength of 1:200,000. This can be doubled for operations on the backbone. Adrenaline can be autoclaved at least once.³⁰

The extent to which a volume of analgesic solution injected into the extradural space will spread is one of the great uncertainties of anaesthesia and because of this it has been suggested that only the serial or continuous injection by means of an indwelling plastic tube, should be employed.³⁵ The spread depends on a number of variables some of them controllable, others not. It is related to the size of the space and this depends somewhat on the build of the patient and on the patency or otherwise of the exits from it, that is from the intervertebral and sacral foramina. It is also related to the rate of absorption of the drug into the blood stream. It is affected by gravity and by the site and speed of injection. The old require less than the young because of the relative impermeability of their

intervertebral foramina. Occasionally the injection of quite a small volume of solution will cause an unexpectedly high block with severe hypotension so it is unwise to use the method unless the patient is judged to be tolerant to moderate hypotension.

The volume of solution injected varies with different workers. For gastrectomy, a tall, fit young man will probably take no harm if 50 ml are injected while 15 ml may well be sufficient for a perineal repair in a small old woman. For a lower abdominal operation, 30-35 ml is an average dose. Each anaesthetist will arrive at what he regards as a proper dosage by experience. One aims not only to get adequate analgesia and relaxation but also to get some hypotension and consequent ischaemia. An injection of 12-15 ml of solution can be said to affect four segments on each side of the point of injection. A rough guide to the average dose may be as follows: for haemorrhoidectomy 15-20 ml, for perineal repair 25-35 ml—hypotension causing ischaemia is often useful in these operations; for herniorrhaphy 25-35 ml; for hysterectomy 30-35 ml, for prostatectomy 15-35 ml; for Caesarean section 25 ml; and for upper abdominal operations 35-50 ml. The addition of one ampoule of hyaluronidase powder to the lignocaine solution is said to hasten the onset of analgesia in sacral extradural blocks, but does not have much effect when the solution is injected from the lumbar area.⁴⁸

Some attention has been given recently to the technique of serial or continuous extradural block, a method first described by Curbelo of Havana in 1949.⁴⁶ He modified Tuohy's technique for continuous spinal analgesia using the latter's needle with a slightly curved point which makes it possible to direct a length of 1 mm bore polyvinyl plastic tubing either upwards or downwards in the extradural space. Through this tubing analgesic solution can be injected at will and so the duration of analgesia can be more or less indefinitely prolonged. The Tuohy needle is rather a spike and because of its size the continuous technique should not be used unnecessarily but in fact, dural puncture with so big a needle rarely happens as with it the loss of resistance test is made very easy and obvious. The tubing should have two or three ink marks made on it with a ball point pen corresponding to the length of the needle. It can be sterilized by boiling and during the process can be kept reasonably free from kinks and bends if it is threaded into a length of polythene tubing. When the Tuohy needle is safely in the space the polyvinyl plastic tube is threaded through it so that at least one and a half needle lengths are in the space. The needle and tubing are now withdrawn together and an inch or two of tubing is left in the space. Whether

delicacy of touch is not used. Mid thoracic puncture is difficult and is perhaps better avoided. If the dura is perforated some workers advise trying again in an adjacent interspace³⁴ but others abandon the method or give an ordinary spinal injection.

The commonly used local analgesic drugs can be employed but lignocaine is rapidly establishing itself as a firm favourite for this as for many other purposes. The solution is stable and can be autoclaved repeatedly. Its effects are rapid in onset—about five to ten minutes—and reasonably prolonged in duration—2–2½ hours using the 1.5 per cent solution. It takes a firm 'bite' on the nerve roots, causes some somnolence following its absorption into the general circulation, and is not very often guilty of causing toxic effects under ordinary clinical conditions. Bromage²⁸ believes in the ability of a 1.2 per cent solution to give a differential block, i.e. sensory analgesia with reflex motor atony but without direct muscular paralysis such as intercostal block. In the writer's opinion 1.5 per cent solution allows very good respiration to take place, while it is rather more certain and sure in action than the weaker solution. A 2 per cent solution seems unnecessarily strong. When a longer period of analgesia is required because of the expected duration of the operation or because prolonged post operative analgesia is thought to be desirable, amethocaine hydrochloride can be added to the lignocaine solution. A convenient way of doing this is to add half the contents of one 100 mg ampoule of crystals to 50 ml of solution giving a final strength of 0.1 per cent. Adrenaline is always used with solutions injected into the extradural space, both to reduce toxicity from rapid absorption into the blood stream and to prolong the effect of the analgesic. 0.25 ml can be added to 50 ml of solution giving a final strength of 1:200,000. This can be doubled for operations on the backbone. Adrenaline can be autoclaved at least once.³⁰

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intervertebral foramina. Occasionally the injection of quite a small volume of solution will cause an unexpectedly high block with severe hypotension so it is unwise to use the method unless the patient is judged to be tolerant to moderate hypotension.

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this is above or below the point of puncture depends on the direction of the curve of the needle. Some workers prefer to have a greater length of tubing inside the space but kinking and curling up of the tubing make this an uncertain manoeuvre and it should be reserved for attempts at true segmental block. The tubing should never be withdrawn through the needle otherwise the distal portion may be sheared off by the sharp edge of the point and left in the extradural space. Solution is then injected from a 5 or 10 ml syringe and a fine needle inserted into the tubing. The syringe is placed in a sterile dressing near the head of the patient and each fresh injection carried out with due regard to asepsis. The method is useful for the maintenance of prolonged sympathetic block e.g. in the lower limbs following injury, operation or thrombosis. Very fine bore neoplex ureteric catheters can be substituted for the polyvinyl plastic tubing.

Conduct of the Anaesthesia

During the operation the patient may be awake and aware of his surroundings, he may be under the influence of an opiate or barbiturate and be sleepy and content. He may be unconscious following the inhalation of gas and oxygen preceded by a small dose of thiopentone or hexobarbitone or he may be lightly anaesthetized with perhaps an endotracheal tube in place. Which of these methods is used depends on the judgement of the anaesthetist and the wishes of the surgeon. A continuous intravenous drip of 500 ml of saline containing pethidine 100 mg and scopolamine 0.4 mg (gr $\frac{1}{15}$) is a useful method of sedation. In upper abdominal operations para-oesophageal vagal block by the surgeon is a useful additional measure. It reduces the frequency of vagal reflexes of which hiccough may be one of the more troublesome.

The maintenance of blood pressure comes second only to the maintenance of proper ventilation and it should be an invariable rule to have an open vein before the block is induced. For this purpose a Mitchell a Gordh a Frankis Evans needle or a Guest cannula can be inserted into a suitable vein or a drip can be set up. With this direct and rapid route to the circulation always available circulatory collapse can almost always be reversed. Breathing is exceptionally easy and free from all trace of bronchospasm during high blocks. This may be due to stimulation of baroreceptors in the aorticocarotid sinuses by hypotension which results in reflex bronchodilatation.²⁷

The indications vary of course with the experience of the anaesthetist and with the preferences of the surgeon and with other

factors. All who use the technique are agreed that the fit patient undergoing an operation below the umbilicus is the most suitable case for the block. It has been successfully used for thoracoplasty³⁸ and recently workers in Canada have been using the method in infants,³⁹ injecting 5-6 ml of 1 per cent lignocaine. Massey Dawkins³⁴ advocates the continuous technique in geriatric patients undergoing such operations as hemicolectomy, gastrectomy, Wertheim's hysterectomy and hernia repairs. He also reports good results in radical mastectomies, injecting 8-10 ml of lignocaine solution between the seventh cervical and the first thoracic spines, with the patient in the lateral position, affected side downwards. In this situation, however, extradural block is not a technique for the beginner.

In therapy, extradural lumbar blocks can be used to ease the severe pain of acute pancreatitis. The relief may be due to relaxation of the sphincter of Oddi. Acute cholecystitis may be similarly treated: the afferent impulses from these viscera entering the cord in the seventh, eighth and ninth posterior roots. Iliofemoral thrombosis has been treated by a continuous block, the catheter remaining in the space up to three days. In such long stay cases penicillin can be added to the solution to reduce the risk of infection.⁴⁰ Pregnancy toxæmia can be successfully relieved,⁴¹⁻⁴³ although the sacral route has certain advantages as well as disadvantages in the treatment of this disorder. Hypertensive heart failure with acute pulmonary oedema is an acute medical condition which has yielded successfully to extradural block.⁴

EXTRADURAL SACRAL (CAUDAL) BLOCK

Although anaesthetists in Britain have not used this interesting technique with the frequency of their United States colleagues it has slowly gained adherents in recent years in surgery, obstetrics and therapy. Now that polyvinyl plastic tubing of 0.015 in. internal diameter and neoplex ureteric catheters can be obtained the continuous technique has been more frequently used than in the past.

The dangers and limitations of the use of the continuous technique in obstetrics have recently been stressed by Hingson.⁴⁴ The primary intra-uterine foetal anoxia when the maternal blood pressure drops below 80 mm of mercury. This is shown by a marked bradycardia, increase in foetal movements and a decrease in liquor amni. A most careful watch must be kept on the maternal blood pressure and pressor drugs must be given if necessary.

this is above or below the point of puncture depends on the direction of the curve of the needle. Some workers prefer to have a greater length of tubing inside the space but kinking and curling up of the tubing make this an uncertain manoeuvre and it should be reserved for attempts at true segmental block. The tubing should never be withdrawn through the needle, otherwise the distal portion may be sheared off by the sharp edge of the point and left in the extradural space. Solution is then injected from a 5 or 10 ml syringe and a fine needle inserted into the tubing. The syringe is placed in a sterile dressing near the head of the patient and each fresh injection carried out with due regard to asepsis. The method is useful for the maintenance of prolonged sympathetic block, e.g. in the lower limbs following injury, operation or thrombosis. Very fine bore neoplex ureteric catheters can be substituted for the polyvinyl plastic tubing.

Conduct of the Anaesthesia

During the operation the patient may be awake and aware of his surroundings, he may be under the influence of an opiate or barbiturate and be sleepy and content, he may be unconscious following the inhalation of gas and oxygen preceded by a small dose of thiopentone or hexobarbitone, or he may be lightly anaesthetized with perhaps an endotracheal tube in place. Which of these methods is used depends on the judgement of the anaesthetist and the wishes of the surgeon. A continuous intravenous drip of 500 ml of saline containing pethidine 100 mg and scopolamine 0.4 mg (gr $\frac{1}{15}$) is a useful method of sedation. In upper abdominal operations parasympathetic vagal block by the surgeon is a useful additional measure. It reduces the frequency of vagal reflexes of which hiccough may be one of the more troublesome.

The maintenance of blood pressure comes second only to the maintenance of proper ventilation and it should be an invariable rule to have an open vein before the block is induced. For this purpose a Mitchell a Gordh a Frankis Evans needle or a Guest cannula can be inserted into a suitable vein or a drip can be set up. With this direct and rapid route to the circulation always available circulatory collapse can almost always be reversed. Breathing is exceptionally easy and free from all trace of bronchospasm during high blocks. This may be due to stimulation of baroreceptors in the aorticocarotid sinuses by hypotension which results in reflex bronchodilatation.³⁷

The indications vary of course with the experience of the anaesthetist and with the preferences of the surgeon and with other

the bowel, interruption of afferent impulses from the pelvic and lower abdominal viscera including the uterus, the birth canal, and denervation of the suprarenal glands

The technique of extradural sacral block is fairly standardized. As in extradural lumbar block, the availability of polyvinyl plastic tubing which can be boiled makes the continuous technique safe and simple. During insertion of the needle into the sacral canal, the gluteal muscles can be put at rest fairly easily in a tense nervous patient if he is told to approximate the big toes while separating the heels as widely as possible. This obliterates the gluteal cleft and makes location of the sacral hiatus easier. In the United Kingdom lignocaine has almost completely displaced procaine, cinchocaine and piperocaine as the analgesic solution of choice. For longer action it can be fortified with amethocaine hydrochloride the 100-mg ampoule of crystals being convenient for this purpose and a strength of 0.15 to 0.2 per cent being suitable.

References

- 1 COPE R W 1954 *Anæsthesia* 9 249 (Oct)
- 2 SANCETTA S M *et al* 1952 *Circulation* 6 559 (Oct)
- 3 DAVIS H and KING W R 1954 *Anæsthesiology* 15 No 6 666 (Nov)
- 4 GRIFFITHS H W C and GILLIES J 1948 *Anæsthesia* 3 134 (Oct)
- 5 GREENE N M 1952 *Surg Gynec Obstet* 95 331 (Sept)
- 6 THORNE T C 1954 *Proc R Soc Med* 47 No 5 301 (May)
- 7 DE CARLE J 1954 *J Amer med Ass* 154 545 (Feb 13)
- 8 MORRIS G *et al* 1954 *Curr Res Anæsth* 33 340 (Sept-Oct)
- 9 PERL J I 1953 *J Int Coll Surg* 20 No 3 343
- 10 DEPP E B 1953 *Ohio St med J* 39 701 (Aug)
- 11 HARALDSON S 1951 *Anæsthesiology* 12 371 (May)
- 12 CAPPE B E *et al* 1953 *Anæsthesiology* 14 398 (July)
- 13 HARRIS J M and HARMEL M II 1953 *Anæsthesiology* 14 390 (July)
- 14 HANSEN H 1953 *Acta chir Scand* 104 481 (April 9)
- 15 GREENE II A *et al* 1954 *Anæsthesiology* 15 No 3 302 (May)
- 16 RENDELL C M 1954 *Anæsthesia* 9 281 (Oct)
- 17 PADDISON R M and ALPERS B J 1954 *Amer med Ass J Neurol Psychol* 71 No 1 87 (Jan)
- 18 CARTER A B *et al* 1954 *Anæsthesiology* 15 No 4 480 (Sept)
- 19 WHITTET T D 1954 *Anæsthesia* 9 No 4 271 (Oct)
- 20 DRIPPS R D and VANDAM L D 1954 *J Amer med Ass* 156 1786 (Dec 12)
- 21 NORMAN J E 1955 *Anæsthesia* 10 No 1 88 (Jan)
- 22 SINCLAIR R N 1954 *Anæsthesia* 9 No 4 286 (Oct)
- 23 ARNER O 1952 *Acta chir Scand Suppl* 167 1-146
- 24 GRADY R W 1954 *Anæsthesiology* 15 No 3 310 (May)
- 25 ROSENBAUM H E *et al* 1952 *Amer med Ass J Neurol Psychol* 68 783 (Dec)
- 26 MAHER R M 1955 *Lancet* 1 II (Jan 1)
- 27 DAWKINS C J MASSEY 1945 *Proc R Soc Med* 38 No 8 299 (April)

they are indicated Autotransfusion of maternal blood by elevation of her legs is a useful auxiliary measure If the block is commenced too early labour may be interrupted or prolonged The posterior position becomes frequent while the forceps rate increases The block should not ordinarily be commenced until the end of stage I when the cervix is 5 cm dilated in primiparae, and 3 cm in multiparae, with effacement of the canal In labours which are expected to be prolonged 0.15 per cent amethocaine solution by intermittent injection through a plastic catheter is advocated while in rapidly progressing labours 1.5 per cent lignocaine or piperocaine through a needle is advised While the perineum is completely relaxed allowing manual rotation and easy forceps delivery, the tone of the abdominal muscles is not so deficient that the patients cannot bear down usefully Loss of blood in the third stage is minimized

Extradural sacral block is not so useful for general surgery as the block given from the lumbar region as more of a potentially toxic agent must be injected to get a comparable result It is however very useful for operations on the anus and perineum and for cystoscopy In the first two conditions post operative pain can be completely relieved if a plastic tube is inserted into the sacral canal through which 10–15 ml of analgesic solution is injected at suitable intervals when the patient is back in bed

In therapeutics the block can give very useful results Obstetricians attest to its usefulness in eclampsia and convulsions can be stopped and the blood pressure lowered to normal limits⁴⁴ In ischaemic disease of the lower limbs the vascular bed in the adult is expanded by an additional 600–800 ml during a caudal block⁴⁵ so that good results are obtained in cases of diabetic gangrene immersion foot and in acute and chronic thrombo embolic conditions The effects of temporary denervation of the vascular supply of the feet and legs can more surely be appreciated after extradural block than after lumbar paravertebral block This is because a bad result after the latter block may be due to failure to deposit the solution near the second and third lumbar paravertebral ganglia whereas uncertainty as to the position of the needle point is rare in sacral blocks

The effects of an extradural sacral or extradural lumbar block up to the eighth thoracic segment are as follows Skin analgesia from the toes to the costal margin; absence of sweating and increase of temperature over the same area due to sympathetic paralysis and dilatation of arteries and veins of the lower limbs muscular relaxation involving the sphincter and the lower limbs the perineal muscles and anterior abdominal wall muscles hyperperistalsis of

CHAPTER 10

REDUCTION OF HÆMORRHAGE DURING OPERATION

Loss of blood during operation is not only a source of danger to the patient but is a major inconvenience to the surgeon. It will therefore be agreed that a bloodless field is desirable if it can be obtained without added risk.

LOCAL METHODS

Local methods of attaining ischæmia have been employed for many years and include the use of rubber bandages and tourniquets to limbs and the infiltration of the operative field with vaso-constrictive solutions such as 1 in 500 000 adrenaline.

Postural ischæmia can sometimes be invoked to provide a dry wound. The principle is a simple one. If a patient is not lying in a perfectly flat and level posture the blood pressure will vary in different parts of his body depending upon the hydrostatic pressure of a column of blood whose length equals the vertical distance between the relevant part and the heart. For example, at heart level the pressure remains constant in any position but elsewhere it will vary on the basis of 2 mm Hg for every inch above or below the heart¹. It is then possible to drop the systolic pressure in the fingers by 40 mm Hg if the arm is elevated to a vertical position (i.e. 20 inches above heart level) and this in itself may diminish bleeding to a very marked extent. In certain parts of the body, postural ischæmia can be used as an adjunct to a very mild degree of induced general hypotension.

INDUCED HYPOTENSION

In pre-anæsthetic days, the excessive psychic and traumatic shock frequently caused fainting at the beginning of an operation. This was welcomed not only by the patient whose agony ceased but also by the surgeon as bleeding was greatly reduced.

The idea of purposely producing a low blood pressure with a view to reducing bleeding from wounds has been developed intensively during the past few years. It has been found that a systolic blood pressure of about 60 mm Hg will assure a practically bloodless field and usually appears to maintain a capillary circulation sufficient for the tissues provided that the blood is fully oxygenated and that

- 28 BROMAGE, P R 1954 *Spinal Epidural Analgesia* Edinburgh and London
E & S Livingstone
- 29 GUTIERREZ A A 1932 *Rev Cirug Buenos Aires* 12 665
- 30 ODOM C B 1936 *Amer J Surg* 34 547
- 31 MACINTOSH R R 1953 *Brit med J* 1 398
- 32 MACINTOSH R. R. 1950 *Anæsthesia* 5 98
- 33 BRUNNER C and IKLÉ, A 1949 *Schweiz med Wochr* 79 799
- 34 DAWKINS C J M 1953 *Anæsthesia* 8 No 4 232 (Oct)
- 35 BRYCE SMITH R 1954 *Anæsthesia* 9 201 (July)
- 36 CURBELO M M 1949 *Curr Res Anæsth* 21 13 (Jan-Feb)
- 37 DALY M and SCHWEITZER A 1952 *J Physiol* 116 35
- 38 DURRANS S F 1947 *Anæsthesia* 2 106
- 39 RUSTON F G 1954 *Canad Anæsth Soc J* 1 37
- 40 THISTLETHWAITE J H *et al* 1953 *Surgery* 33 818
- 41 OSTLERE G 1952, *Anæsthesia* 7 No 3 169 (July)
- 42 SARNOFF S J and FARR H W 1944 *Anesthesiology* 5 69
- 43 HINGSON R A 1954 *Anesthesiology* ed Donald Hale ch 19 Phila
delphia F A Davis
- 44 LEVENE, W., *et al* 1944 *Amer J Surg* 64 3
- 45 BRYCE SMITH R and WILLIAMS E O 1955 *Lancet* 2 1241
- 46 ROSSER, B H SCHNEIDER M 1956 *Anesthesiology* 17 288
- 47 PAYNE, J P BERGENTZ, S E 1956 *Lancet* 1 666 (May 11)
- 48 SCOTT D B 1956 *Brit J Anæsth* 28 187
- 49 WALSH F 1956 *Lancet* 1 859 (June 2)
- 50 THOMAS D V 1956 *Anesthesiology* 17 752 (Sept)

Arteriotomy

This technique is founded on experimental work in animals in which the effects of bleeding through an arterial cannula were studied.⁴ It was found that it was possible to maintain a state of extreme hypotension for 135 minutes and if at the end of this time the blood was returned through the same cannula, recovery was more rapid than if an intravenous retransfusion was given. The method was first used in man to control blood loss in cerebral surgery,⁵ and various types of apparatus have been devised for the purpose.⁶ The one illustrated has proved most successful in the neurosurgical department of St Bartholomew's Hospital.⁷ It is clear that this method is simply one of inducing a controlled degree of hæmorrhagic shock and is accompanied by vasoconstriction. This may be so severe that a brachial plexus block may be necessary to inhibit the arteriospasm and enable the ready withdrawal and return of blood to continue.⁸ In view of the importance which has been attached to vaso dilatation in conjunction with hypotension it is curious that such good results have been obtained with arteriotomy. However the technical difficulties and complications inseparable from this method have led to its virtual abandonment in this country and it will not be discussed further.

Drugs which Block Autonomic Ganglia

The autonomic ganglia can be blocked by various drugs those which have been investigated most fully being tetraethylammonium chloride and the methonium and thiophanium compounds. Since these drugs have an unselective action, the effect produced is that of sympathetic and parasympathetic paralysis unlike the mainly sympathetic paralysis produced by spinal blocks. The chief side actions on conscious patients are dryness of the mouth paralysis of accommodation gut distension (occasionally progressing to paralytic ileus) and difficulty in micturition. Since the blood pressure can be controlled to an appreciable extent by posture, it is practically certain that the hypotension is caused by loss of vasomotor tone.⁹ Although the hypotensive effect of tetraethylammonium chloride was first described in 1914¹⁰ it has never been popular owing to its transient effects and various side actions.

The methonium compounds are quaternary ammonium substances with very low lipid solubility so that they probably become attached to the surface of the cells upon which they act but do not actually enter them. Their common formula is $(\text{CH}_3)_3\text{N}(\text{CH}_2)_n(\text{CH}_3)_3$ the n being the variable

vaso dilatation is ensured. How far this assumption is justified is considered later in the chapter. So far, three methods of inducing hypotension have been employed

Spinal Block

Total spinal block (intrathecal) is considered in Chapter 9. As most of the sympathetic system is paralysed the blood pressure is largely dependent on the patient's position and this constitutes the basis of control.²

High extradural block. If a block is carried up to the level of T 3 the systolic pressure usually falls to between 60 and 80 mm Hg whatever was the initial pressure.³ This effect is again caused by pre ganglionic sympathetic paralysis and is accompanied by vaso-dilatation. See also Chapter 9.

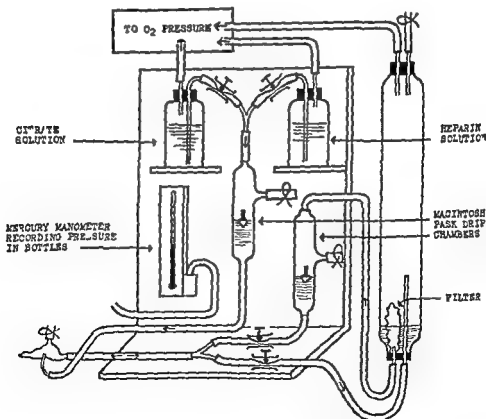


FIG 57 Jackson's apparatus for arteriotomy (*Anæsthesia*)
(Modified from original design by P. L. F. Mortimer)

resulted in a 12 per cent reduction of cerebral blood flow while the oxygen consumption remained unaltered. This resulted in symptoms of hypoxia.¹⁸ Under general anaesthesia, however, there is diminished cerebral oxygen consumption so that there is less risk of hypoxia with mild degrees of hypotension. If there is any atheromatous change in the carotid arteries it is said that any severe fall in systolic pressure is likely to produce a catastrophic necrosis of a large part of the area supplied by the middle cerebral artery.¹⁸ A death from this cause following hypotension induced by pentamethonium bromide has been described.¹⁹ Hypoglycaemia tends to follow the administration of the methonium compounds and it has been shown that the brain is very intolerant of this condition if hypotension is present.²⁰ In diabetics, the blood sugar, already reduced by the methonium compounds, may fall still lower when insulin is given.⁴¹ Severe hypoglycaemia may result and this may be indicated during anaesthesia by a progressive tachycardia. Sudden death has occurred during induced hypotension and is usually due to coronary insufficiency. It is unfortunate that coronary disease occurs with increasing frequency in young patients who are apparently healthy and show normal E C Gs. The condition may therefore be virtually impossible to detect⁴¹ and it is in patients suffering from it that the chief danger lies. The coronary blood flow is determined mainly by the aortic pressure,⁴⁰ so that severe hypotension may reduce the flow to the point at which cardiac failure occurs.⁴¹

Opinions still differ as to the relationship of induced hypotension and shock. It has been stated that since it is difficult to produce irreversible shock in sympathectomized dogs autonomic block with the methonium drugs will afford similar protection. In the writers' opinion this argument is unsound and in practice severe haemorrhage occurring in a hypotensive patient can very rapidly cause death. On the other hand in established shock which has not responded to efficient treatment, the injection of a methonium compound might possibly cause a more favourable distribution of the blood without lowering the pressure much further¹ and by relieving vaso spasm might enable a rapid fluid infusion to be given.

The excretion of penta and hexa methonium halides seems to be almost entirely through the kidneys the actual mechanism being mainly glomerular filtration with minimal tubular excretion.²² While a moderate degree of induced hypotension with the methonium drugs does not appear to impair renal function to an appreciable extent²³ yet in volunteers the glomerular filtration rate and urine flow dropped sharply and this prolongs the action of the drugs.²⁴

Pentamethonium (C 5) $\left\{ \begin{array}{l} \text{bromide (lytensium)} \\ \text{iodide (antilusin)} \end{array} \right\}$ was originally introduced as an antidote to the muscle relaxant decamethonium (q v) but was soon found to cause a marked fall in blood pressure. It is put up in 2.5 ml ampoules containing 50 mg of the drug.

Hexamethonium (C 6) $\left\{ \begin{array}{l} \text{bromide (vegolysen)} \\ \text{iodide (hexathide)} \end{array} \right\}$ has a very similar action to pentamethonium but is thought by some observers to have a more constant effect.¹¹

Various techniques have been used, an initial intravenous dose of 20 mg of either drug being common, subsequent injections, after a wait of at least three minutes, being given to maintain the systolic pressure at about 60 mm Hg. If possible, a head up tilt is maintained. It has been pointed out that young healthy adults tend to develop tachycardia with little hypotension and thus require a larger dose and steeper anti-Trendelenburg tilt than older ones, especially those with arteriosclerosis who show little change in pulse rate but marked hypotension.¹ Some workers believe in giving the comparatively large initial dose of 50 mg to healthy adults, but the technique cannot be regarded as safe. If a reasonable dose of a methonium drug does not produce sufficient effect, a supplementary intravenous injection of from 0.2 to 1 g procaine amide hydrochloride (procaryl, pronestyl) can be given slowly. This drug not only increases the hypotension but usually slows the pulse.¹² A Gordh needle or one of its many modifications is useful for these multiple injections.

A great deal of work has been carried out recently on the effects of the methonium drugs on the brain. The recovery of consciousness is slightly delayed and study of electro-encephalograms suggest that this is probably due to stagnant hypoxia of the cerebral cortex.¹⁴ It is likely that the hypoxia may be partially compensated for by the increased oxygen extraction (A-V O₂ difference) which occurs when the blood flow is slowed down.¹⁵ Fifteen patients in whom hypotension had been induced by hexamethonium were investigated post operatively and six showed a positive flicker fusion intensity test.¹⁶ This was probably due to hypoxia which could undoubtedly lead to irreversible damage.¹⁷ Although experimental work on the cerebral blood flow is complicated by the varied oxygen consumption in the conscious and anaesthetized states it seems probable that from 55 to 60 mm Hg is the critical blood pressure in healthy patients and it should not be allowed to fall below this point. In conscious hypertensives a 26 per cent reduction in mean arterial pressure

resulted in a 12 per cent reduction of cerebral blood flow while the oxygen consumption remained unaltered. This resulted in symptoms of hypoxia³⁸. Under general anaesthesia, however, there is diminished cerebral oxygen consumption so that there is less risk of hypoxia with mild degrees of hypotension. If there is any atheromatous change in the carotid arteries it is said that any severe fall in systolic pressure is likely to produce a catastrophic necrosis of a large part of the area supplied by the middle cerebral artery³⁹. A death from this cause following hypotension induced by pentamethonium bromide has been described¹⁸. Hypoglycaemia tends to follow the administration of the methonium compounds and it has been shown that the brain is very intolerant of this condition if hypotension is present⁹. In diabetics, the blood sugar, already reduced by the methonium compounds, may fall still lower when insulin is given⁴⁰. Severe hypoglycaemia may result and this may be indicated during anaesthesia by a progressive tachycardia. Sudden death has occurred during induced hypotension and is usually due to coronary insufficiency. It is unfortunate that coronary disease occurs with increasing frequency in young patients who are apparently healthy and show normal ECGs. The condition may therefore be virtually impossible to detect⁴¹ and it is in patients suffering from it that the chief danger lies. The coronary blood flow is determined mainly by the aortic pressure⁴⁰ so that severe hypotension may reduce the flow to the point at which cardiac failure occurs⁴¹.

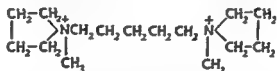
Opinions still differ as to the relationship of induced hypotension and shock. It has been stated that since it is difficult to produce irreversible shock in sympathectomized dogs, autonomic block with the methonium drugs will afford similar protection. In the writers' opinion this argument is unsound and in practice severe haemorrhage occurring in a hypotensive patient can very rapidly cause death. On the other hand in established shock which has not responded to efficient treatment, the injection of a methonium compound might possibly cause a more favourable distribution of the blood without lowering the pressure much further²¹ and by relieving vaso spasm might enable a rapid fluid infusion to be given.

The excretion of penta- and hexa methonium halides seems to be almost entirely through the kidneys, the actual mechanism being mainly glomerular filtration with minimal tubular excretion²². While a moderate degree of induced hypotension with the methonium drugs does not appear to impair renal function to an appreciable extent²³ yet in volunteers the glomerular filtration rate and urine flow dropped sharply and this prolongs the action of the drugs²⁴.

In patients with diseased kidneys, methonium hypotension may accelerate the rise in blood urea and precipitate uræmia.²⁵ Even if this does not occur the renal function may be further impaired.²⁶

Effects on the liver have not yet been fully worked out, but it has been observed that if the blood pressure drops below 60 mm Hg, this organ becomes cyanosed, turgid and rubbery, indicating some degree of anoxia.²⁷ It seems certain that occult liver damage can result as the ability to clear bromsulphalein may be impaired,²⁸ and fatal necrosis has been reported.²⁸

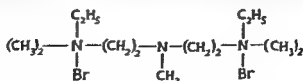
Pentolinium tartrate The drug known as pentolinium tartrate (ansolisen, M and B 2050A) is pentamethylene 1.5 bis (1 methyl pyrrolidinium) hydrogen tartrate. Although not a methonium



Pentolinium

compound its pharmacological effects seem similar. It has been used as a 0.5 per cent. aqueous solution 10 to 20 mg. being given intravenously to fit adults below 50 years of age 3 to 5 minutes before operation. The maximum fall in blood pressure occurs in about 3 minutes and the operating table tilt is then adjusted until the systolic pressure measured at heart level is stabilized at about 65 mm Hg.²⁹ Tachycardia is said to be rare unless chlorpromazine has been included in the anaesthetic technique.

Azamethonium bromide (pendiomide, pentamethazine, Ciba 9295) is pentamethyl diethyl 3-azapentane 1.5-diammonium dibromide



It appears to be more controllable than the methonium compounds but less so than trimetaphan. The average dose necessary lies between 50 and 100 mg. given at the rate of 10 mg. per minute.

The drug has had some vogue in Germany.⁴³

Trimetaphan camphorsulphonate (arfonad, RO-2-2222) is a thio-phanium derivative being d.3.4 (1.3-dibenzyl 2-keto-imidazolido)

1,2 trimethylene thiophanium-d-camphor sulphonate³⁰ It differs from the methonium compounds in being eliminated rapidly and this enables it to be given as a 0.1 per cent intravenous drip infusion. In children a 0.2 per cent solution may be advisable to minimize the risk of overloading the circulation.³¹ Even stronger solutions are possible if a large electrically driven syringe is employed but this is still in the experimental stage.³ If the usual 0.1 per cent trimetaphan solution is used an initial rate of 50 to 60 drops per minute is tried and then adjusted to give the required blood pressure. Discontinuance of the infusion should result in the speedy restoration of the pressure to its previous level. This technique is certainly more controllable than single injections of the methonium drugs and has been used successfully in neurosurgery.³² If for any reason a drip infusion is impracticable trimetaphan can be given in fractional doses through a Gordh's needle using the 50 mg per ml solution supplied in ampoules.³⁴ An initial adult dose of from 10 to 50 mg followed by repeated doses of from 10 to 30 mg at 10- to 15 minute intervals can be employed to maintain the systolic pressure at between 60 and 80 mm Hg but the pressure chart will of course be much more irregular than with a drip infusion.

Trimetaphan forms a white precipitate with thiopentone and with gallamine and is incompatible with bromides and iodides. It may like pethidine produce weals. There is some evidence that it has a local dilating action on vessel walls in addition to its ganglion blocking effects. Its hypotensive action is potentiated by deep narcosis and by ether. About one third of the drug infused is recoverable from the urine within a few hours, but the fate of the remainder is at present unknown. The brief duration of activity is also unexplained. Occasional renal failure some days after operation is a disquieting sequela and the drug is said to liberate histamine.

Negative Pressure to Limbs

An ingenious adjunct to any method of controlled hypotension has recently been evolved. It is well known that compression of the legs and thighs e.g. by tight bandaging raises the systemic blood pressure and conversely the application of negative pressure lowers it. A metal cylinder encloses each of the patient's legs, a seal at the top of each thigh being made with a rubber diaphragm. After a moderate fall of pressure has been induced a vacuum of from 30 to 40 mm Hg is applied to the legs by means of a powerful suction pump and this usually ensures a further pressure drop of about 30 mm Hg irrespective of posture. As the air pressure can be

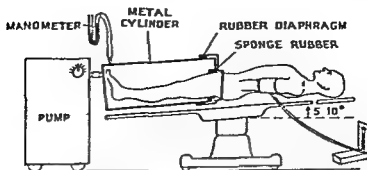


FIG 58 Negative pressure device showing application of cylinders to patient's leg. (J W Saunders *Lancet*)

varied at will between wide limits this technique probably reduces some of the risks of induced hypotension³⁵

A series of 150 neurosurgical operations has been reported using the modified apparatus shown³⁶

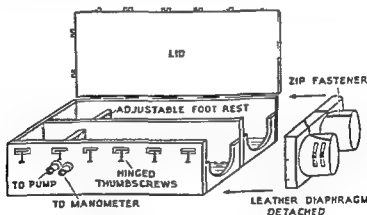


FIG 59 Negative-pressure apparatus modified for neurosurgery (A James *et al.*, *Lancet*)

Anæsthetic Technique

When using induced hypotension the anæsthetist must remember that he has converted a comparatively fit patient into a temporarily shocked one and he will consequently require a greatly reduced dosage of narcotic drugs. Furthermore the reduced blood flow through vital organs makes it imperative that full oxygenation be ensured or some degree of hypoxia will occur. Controlled breathing and an open peritoneal cavity both increase the hypotension to a slight degree. Measurement of the blood pressure by means of an oscillometer is highly recommended by anæsthetists who are experienced in hypotensive techniques.

Great care is necessary if cuffed tracheal tubes are used as the pressure within them may exceed the reduced systolic pressure with consequent necrosis or ulceration of the mucosa³⁷

Post-operative Care

At the end of operation with the table level the patient's systolic pressure should be about 100 mm Hg. If much below this figure a head down tilt should be adopted and if necessary a pressor drug should be given. Methedrine 10 mg is probably as effective as any other but unfortunately its effect is evanescent and it may cause restlessness during the recovery stage. It is most important that the patient's head should not be raised in transit to the ward and a tilting trolley is invaluable. Constant supervision is essential until the blood pressure has become stabilized at a satisfactory level.

Morbidity and Mortality

Sudden death occurring during an operation conducted under induced hypotension is usually due to cardiac failure from the fall of pressure in the coronary arteries as discussed under the action of the methonium compounds. Delayed deaths may occur from cerebral thrombosis and renal failure.

In an endeavour to assess the risk to life associated with induced hypotension a questionnaire was sent to 602 anaesthetists in Great Britain⁴. 21 000 cases were reported with 46 deaths a mortality rate of 1 in 459.

The main non fatal complications seem to be associated with cerebral hypoxia and, apart from obvious disasters such as occasional blindness⁴³ are characterized by changed mental attitude and deterioration of ability of the patient.

It is possible that if the metabolic needs of the vital organs can be diminished without added risk by methods such as induced hypothermia (see Chapter 12) hypotension deliberately produced would have a materially lessened morbidity.

In operations conducted under induced hypotension it is essential for the surgeon to secure even the smallest blood vessels. Otherwise the incidence of reactionary hæmorrhage when the pressure becomes normal will be increased.

Indications

From a consideration of the preceding facts it must be inferred that at the present time induced hypotension should be reserved for patients suffering from a fatal disease or some gross disability for

the relief of which an operation is necessary which cannot be undertaken unless a bloodless field is provided

In the writers opinion the technique is unjustified if it merely renders a surgeon's work easier in an elective operation. On the other hand, it may happen that an anæsthetist is urged by his surgeon to reduce the patient's blood pressure during such operations as fenestration, dacryorhinostomy, mammoplasty, etc., and if the greatest possible care is taken the operating conditions can be very rewarding

Contra-indications

Induced hypotension appears to be specially dangerous in the following conditions —

Angina and any state suggestive of coronary insufficiency

Previous cerebral thrombosis

Addison's disease and cachectic shocked and aged patients (but see above)

Diabetes in which condition severe hypoglycæmia may occur

Chronic hypertension associated with pulmonary or renal disease

Late stages of pregnancy in which foetal hypoxia may ensue

It is quite remarkable how bleeding can be reduced if proper attention is paid to the principles of good anæsthesia. These will include a smooth induction maintenance of a perfect airway avoidance of coughing and straining appropriate posture adequate ventilation with the avoidance of hypercarbia and hypoxia the use of non rebreathing valves open circuits etc

References

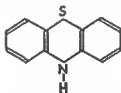
- 1 ENDERBY G E H 1954 *Lancet* Jan 23 185
- 2 GRIFFITHS H W C and GILLIES J 1948 *Anæsthesia* Oct 134
- 3 BROMAGE P R 1951 *Anæsthesia* Jan 26
- 4 KOHLSTAEDT K G and PAGE, I H 1943 *Arch Surg* 47 178
- 5 GARDNER W J 1946 *J Amer med Ass* 132 572
- 6 HALE, D E. 1948 *Anesthesiology* 9 498
- 7 JACKSON L. 1954 *Anæsthesia* Jan 13
- 8 MORTIMER, P L F 1951 *Anæsthesia* July 128
- 9 { PATON W D M 1951 *Brit med J* 1 773
- 9 { McMICHAEL, J 1952 *Proc R. Soc Med (Sec Med)* Feb 26 (discussion)
- 10 MARSHALL, C R. 1914 *Trans R Soc Edinb* 50 379
- 11 SHACKLETON R. P W 1951 *Brit med J* May 12 1054
- 12 { ENDERBY G E H and PELMORE J F 1951 *Lancet* March 24 663
- 12 { HUGHES S 1951 *Lancet* March 29 666
- 13 { MASON A. A. and PELMORE, J F 1953 *Brit med J* Jan 31 250
- 13 { ASERMAN D 1953 *Brit med J* May 2 961

- 14 BROMAGE, P R 1953 *Proc R Soc Med* (An Sec.) May 1
- 15 MORRIS G C., and MOYER J H 1954 *Lancet* July 31 246
- 16 BERG O *Acta psychiat Abh suppl* 58
- 17 NILSON E. 1953 *Brit J Anaesth* Jan 24
- 18 DENNY BROWN D L. 1952, *New Engl J Med* 246 839
- 19 BODMAN R I 1952 *Lancet* Nov 21 1085
- 20 GRIFFITHS J A 1953 *Quart J Med* 22 405
- 21 WYMAN J B 1953 *Proc R Soc Med* (Secs Surg and An) Feb 4
- 22 YOUNG I M *et al* 1951 *Brit med J* Dec. 22 1500
- 23 EVANS B and ENDERBY G E. H 1952 *Lancet* May 24 1095
- 24 { McQUINN E. G 1952 *Med J Aust* 1 769
- 25 { MACKINNON J 1952 *Lancet* July 5 12,
- 26 ROSENHEIM M L. 1952 *Proc R Soc Med* (Sec Med) Feb 26
- 27 BLANEY J D 1952, *Lancet* May 17 990
- 28 BROMAGE, P R. 1952 *Lancet* July 5 10
- 29 LYNN R. B *et al* 1952 *Surgery* 32 211
- 29 ENDERBY G E. H 1954 *Lancet* Nov 27 1097
- 30 SADOVE, M S *et al* 1953 *Anaesthesia* July 175
- 31 ANDERSON S 1955 *Brit med J* July 9 103
- 32 BOWEN R. 1955 personal communication.
- 33 ANDERSON S and McILISOCK W 1953 *Lancet* Oct 10 754
- 34 KILDUFF C J 1954 *Lancet* Feb 13 337
- 35 SAUNDERS J W., 1952 *Lancet* June 28 1286
- 36 JAMES A *et al* 1953 *Lancet* Feb 23 412
- 37 BELAN O H and ZUCK D 1953 *Anaesthesia* April 96
- 38 KETY S S *et al* 1950 *J clin Invest* 29 402
- 39 BLUMGART H L *et al* 1940 *Amer Heart J* 19 1
- 40 ECKENHOFF J E. *et al* 1947 *Amer J Physiol* 148 582.
- 41 HAYWARD G 1952 *Anaesthesia* April 67
- 42 HAMPTON L. J and LITTLE D M 1953 *Lancet* June 27 1299
- 43 GOLDSWORTH A J B and HEWER A J H 1952 *Brit med J* Oct 4 759
- 44 GRIFFITH J A 1953 *Quart J Med* Oct 405
- 45 KIRSCH J *Med Klinik Berlin* 1953 48 1516

CHAPTER 11

THE PHENOTHIAZINE DERIVATIVES

A GREAT deal of interest has been shown recently in the derivatives of phenothiazine which has the structural formula —



These compounds were originally termed antihistamines but since chlorpromazine one of the most useful members has little antihistaminic action the term should be avoided. They mostly belong to a somewhat vague group of drugs known as chemical tranquilizers. The most familiar phenothiazine derivatives are the five listed below. Their structural formulae are shown opposite.

Pacatal (P 391) N methyl piperidyl 3 methylphenothiazine

Ethopropazine hydrochloride (lysivane)

Diethazine hydrochloride (antipar, diparcol, latibon, R P 2987)

3 diethylaminoethyl N phenothiazine HCl

Promethazine hydrochloride (atosil, fargan, phenergan) Avomine is promethazine 8 chlorothiophyllinate) N 2 dimethylamino-N propylphenothiazine HCl

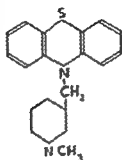
Chlorpromazine hydrochloride (ampliactil, amplictil, hibernol, largactil, M & B 2378, megaphen, R P 4560, thorazine, 2601A S K F) 3 chloro 10 (3 dimethylaminopropyl) phenothiazine HCl

A brief summary of some of the pharmacological actions of four of these compounds is given in the appended table ¹⁸

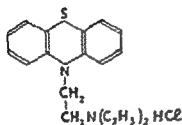
	Promethazine	Diethazine	Chlorpromazine	Pacatal
Antihistaminic	+++	+	+	++
Parasympatheticolytic	++	+	+	++
Sympatheticolytic	+	+++	+++	++
Hypotensive	+	+	++	++
Hypothermic	+	++	+++	+

Since chlorpromazine seems to be the drug of most interest to anaesthetists a short account must be given of its effects. It should

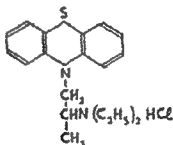
be admitted however, that although it is some years since it was first described by French workers¹ its pharmacology still remains obscure. This may be due to the fact that the action on animals and man is by no means identical.



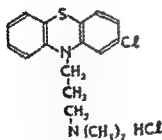
Pacatal



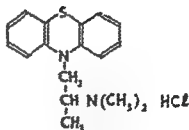
Ethopropazine



Diethazine



Chlorpromazine



Promethazine

Cardiovascular System There is little effect on blood pressure although it tends to fall slightly and to become orthostatic. If hypotension should occur it is probably due to peripheral vasodilatation and the correct treatment is by adopting a head-down tilt and not by the administration of pressor amines as their effects may be inverted.² The pulse rate tends to rise but there is no

significant change in the electrocardiogram except occasional sinus arrhythmia. In arterial blood there is no change in the pH, sodium, potassium, chloride, or phosphate levels, nor in the oxygen, carbon dioxide, haemoglobin or haematocrit values. The blood sugar tends to rise and the urea nitrogen to fall. After prolonged administration agranulocytosis can occur sometimes with fatal results² and myocardial depression eventually takes place.

Central and Autonomic Nervous Systems Chlorpromazine is not a true analgesic but seems to render patients more indifferent to pain which they tend to regard objectively. On the other hand the statement that the drug disconnects the cortex from the diencephalon thus producing a chemical leucotomy does not appear to rest on any solid foundation. It would probably be nearer the mark to say that chlorpromazine appears to act in a considerable degree on the diencephalon so that it induces a partial chemical hypothalamectomy⁴. The drug shows a powerful antagonism to adrenaline⁵ but less to noradrenaline⁶, histamine, acetylcholine or cholinesterase⁷. It was originally stated to produce a complete autonomic block, i.e. that it was a "ganglioplégique" but later work has shown it to possess little if any ganglion blocking activity⁸. It thus resembles dibenamine, phentolamine and other adrenolytic compounds⁹. The fall in body temperature which may take place after chlorpromazine has been given was at first thought to be due to autonomic block but is now considered to be partly the effect of a depressant action on skeletal muscle. The specific adrenolytic action of chlorpromazine probably accounts for its ability to protect to a certain extent against traumatic shock. The theory that this effect was due to the prevention of A.C.T.H. release has now been disproved so that the idea of the drug causing a chemical adrenalectomy or chemical hypophysectomy⁷ would seem to be a fallacy¹⁰. A French biochemist¹¹ has studied the effects of chlorpromazine on many species of vegetables and animals and regards it as having a unique mode of action which he has named "narcobiotique". In small doses the cerebral action of the compound closely resembles that of general anaesthetics but larger doses do not produce anaesthesia, and furthermore, chlorpromazine does not appear to affect oxidative processes directly as anaerobic organisms are susceptible to its action. It is suggested that it prevents the effects of sympathetic irritation which normally set in motion the "pituitary-adrenal mechanism"¹² by a depressant action on the reticular formations of the brain combined with peripheral antagonism to adrenaline. These reticular formations are believed to contain centres which control vomiting,

heat regulation, wakefulness, muscle and vasomotor tone and the secretion of the anterior lobe of the pituitary gland. The general depression of these functions might explain the diversity of actions claimed for chlorpromazine.¹¹ The electro-encephalogram usually shows a normal sleep pattern.¹⁴

Respiratory System The tidal volume is lessened owing to central depression although the respiratory rate is sometimes raised. In spite of this depressed ventilation the oxygen consumption tends to be raised.

Gastro intestinal System Chlorpromazine reduces gastric secretions and has an anti spasmotic effect on the small intestine. Post-operative nausea and vomiting appear to be significantly reduced.

The Liver Chlorpromazine has a definitely toxic action on the liver¹⁵ and frequently causes slight jaundice which is of the obstructive type. If given over a prolonged period, e.g. in psychiatry, this effect may be serious.

Excretion The excretion of the drug is slow, only a small amount being recoverable from the urine. No effective antagonist is known at the time of writing.

Physical Properties Chlorpromazine is a white crystalline powder with a slight smell. Its molecular weight is 355.3 and its melting point 195° C. It is readily soluble in water and is usually put up in ampoules of either 1 per cent. or 2.5 per cent. solution. The pH of the latter solution is about 5.5 and the addition of alkaline fluids (such as sodium barbiturates) will cause precipitation. The acidity of chlorpromazine solution makes it irritating and consequently it should never be given subcutaneously and intravenous injections should be well diluted. It is possible to give the drug by deep intramuscular injection but it should be kept well away from any important nerve. Skin sensitization occurs rapidly in some people in which case irritating rashes can develop. Chlorpromazine can also be supplied as sugar-coated tablets of 25 mg. and 10 mg. for oral use and rectal suppositories can be obtained.

Clinical Uses In anæsthetic practice chlorpromazine can usefully be employed for the following purposes—Firstly, before and after operation it is a sedative in the sense that it produces a feeling of detachment and indifference to pain although it is not a true analgesic. Secondly, it reduces post operative vomiting not due to mechanical causes.¹⁷ Thirdly, it probably affords some protection against the onset of shock in prolonged and traumatic operations. It is however, doubtful if it does more good than harm in established shock owing to peripheral vaso-dilatation. Fourth, it depresses

significant change in the electrocardiogram except occasional sinus arrhythmia. In arterial blood there is no change in the pH, sodium potassium chloride, or phosphate levels, nor in the oxygen carbon dioxide, haemoglobin or haematocrit values. The blood sugar tends to rise and the urea nitrogen to fall. After prolonged administration agranulocytosis can occur sometimes with fatal results³ and myocardial depression eventually takes place.

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- 9 HULDEBRÖ I., 1954 *Arch int pharmacodyn* 98 309
- 10 ARON E. *et al* 1953 *Bull Acad nat Méd Paris* 137 417
- 11 DECOURT P. L. 1953 *Thérapie* 8 846
- 12 SELYT H. 1952 *The Story of the Adaptation Syndrome* (8 (Montreal)
- 13 HOPKIN D. A. B. 1955 *Lancet* March 19 405
- 14 BEARD A. J. W. 1953 *Proc R Soc Med (An Sec)* Dec 4
- 15 Annotation, 1955 *Brit med J* Feb 5 338
- 16 ALLUAUME E. 1952, *Anésth & Analges* 9 261
- 17 BOULTON T. 1955 *Anaesthesia* July 233
- 18 DAVIES J. I. HUGGINS D. H. M. and WOLKENSTEIN C. I. 1956 *Canad Anesth Soc J* July 224

respiratory reflexes facilitating intubation and often terminating an attack of hiccups. Lastly, by the prevention of shivering it facilitates the induction of hypothermia (q v) if this is considered to be desirable.

Administration and Dosage: Chlorpromazine can be given by mouth in doses of about 50 mg the night before operation and again in the last drink. Alternatively, for premedication a rectal suppository incorporating about 200 mg can be used in a fairly fit adult. If a more rapid effect is desired, about 50 mg can be given by intravenous injection but it is essential that a diluted solution should be used as the contents of an original ampoule should be diluted to at least 20 ml and injected very slowly either directly into a vein or preferably into the tubing of a drip infusion if such is already set up. It will be found that a reduction in dosage of subsequent inhalation or intravenous anaesthetics and muscle relaxants can be effected but it is still uncertain whether true potentiation or 'synergism' occurs or whether the effect is one of simple summation of the actions of chlorpromazine with those of the other drugs. If small amounts of chlorpromazine are given after operation the usual post operative analgesic drugs can be drastically curtailed or even omitted altogether. It is wise to substitute pethidine and scopolamine for the more usual omnipon-scopolamine as a premedication injection if chlorpromazine is in use owing to the risk of increasing the anti diuretic effect of morphine and its various derivatives.¹⁶

All kinds of combinations of chlorpromazine with other drugs have been described somewhat unscientifically as 'cocktails', 'mélanges' and 'soups' and it is quite impossible even to enumerate these. The most popular lytic cocktail at the time of writing is a mixture of pethidine 100 mg in 2 ml chlorpromazine 50 mg in 2 ml promethazine 50 mg in 2 ml. This is either diluted and injected very slowly into a vein until the desired effect is obtained, or it is given as a drip infusion.

References

- 1 { LABORIT H and HUGUENARD P 1952 *Pr méd* 60 1455
- 2 { LABORIT H and HUGUENARD P 1954 *Pratique de l'hibernothérapie* Paris
- 3 DOBKIN A II *et al* 1954 *Anaesthesia* July 157
- 4 TASKER J R 1955 *Brit med J* April 16 950
- 5 PARFITT D N 1955 *Brit med J* April 30 1094
- 6 KOPERA J and ARMITAGE A K 1954 *Brit J Pharmacol* 9 392
- 7 FOSTER C A *et al* 1954 *Lancet* 2 614
- 8 COURVOISIER S *et al* 1953 *Arch int pharmacodyn* 92 305
- 9 HOLZBAUER M and VOGT H 1954 *Brit J Pharmacol* 9 402

was shown in Edinburgh in 1947²² Following this lead, surgeons and physiologists throughout the world embarked on a new sea of adventure, and reports followed from Laborit and Huguenard, 1951,⁴ who used their lytic cocktail to produce some hypothermia, from Juvenelle, 1952,⁶ Delorme, 1952,⁷ Lewis and Taufic, 1953⁸, Swan *et al*, 1953,⁹ Dundee, Gray and their colleagues 1953¹⁰ Boerema *et al*, 1951¹¹ and from Ross, 1954¹² and Scurr in 1955³⁰ The chief surgical uses of hypothermia are for interruption of the circulation in operations on the brain the heart and the abdominal aorta

Clinical hypothermia has been classified into light intermediate and deep by Delorme³ Light hypothermia with temperatures between 35 and 30° C has been employed in asphyxia neonatorum,⁴¹ acute poliomyelitis associated with hyperpyrexia and bulbar paralysis⁴² post partum hæmorrhage with irreversible shock⁴³ to increase the safety of hypotension during operations on the brain, and later as a therapeutic measure to allow the brain to survive with a lowered metabolism⁴⁴ acute hyperthyroidism tetanus and following acute hypoxic episodes Light anaesthesia with the use of relaxants to prevent shivering, together with exposure to a cold environment are all that is required although chlorpromazine has been used by some workers because it too inhibits shivering and aids vaso-dilatation Intermediate hypothermia in which the temperature is lowered to 28 to 25° C is the method used for interrupting the blood flow to the brain heart liver or kidneys for periods varying from five to fifteen minutes Deep hypothermia with temperatures between 20 and 15° C is still a laboratory adventure It has been possible to cool dogs to the level of spontaneous arrest of the heart (18–12° C) and to resuscitate them successfully by rewarming

Effects of Hypothermia

These are most ably dealt with by Delorme, 1955¹³ and by Churchill Davidson⁴⁵ In experiments on dogs it has been shown that cooling and rewarming between 38° C and 25° C by the pervascular or extracorporeal method is harmless but that ventricular fibrillation is a real danger if the heart is manipulated or incised under hypothermic conditions and moreover only two thirds of such cases of fibrillation can be reversed by electrical means Oxygen consumption may show an initial rise even while the temperature is falling but soon decreases proportionally to the fall in temperature Renal ischaemia for four or five hours under hypothermia does not lead to uræmia although the mean effective renal blood flow decreases to approximately half of normal at a temperature of

CHAPTER 12

INDUCED HYPOTHERMIA

History—Effects of Hypothermia—Methods of Producing Hypothermia—Re warming—Methods of Anaesthesia during Hypothermia—Complications—Uses of Induced Hypothermia

IN an effort to decrease the demand for oxygen by the cardiac and cerebral cells during periods of decreased blood flow in surgical operations resort has to be made to artificial cooling and the hypometabolism which results from it. Cooling enables the cells of the brain, parenchymatous organs and myocardium to withstand periods of hypoxia during failure of oxygen supply which in its absence would prove injurious and might end fatally. It also prevents hypoxia from wrecking the machinery by postponing the secondary enzymatic changes which in normal subjects accompany this insult. It has been said that perivascular cooling slows the machine by easing up on the throttle and not by increasing the load.² For intracardiac operations the adult patient must be cooled to approximately 30–28° C, the child patient to 26–24° C and at this temperature both heart and brain can be excluded from the circulation for about ten minutes.

History While cooling for purposes of anaesthesia has been in vogue at least since the Napoleonic era, cooling for use in cardiac surgery to provide hypometabolism dates from the work of Bigelow of Toronto in 1950.^{1, 39} His predecessors in experiments on cooling included Simpson and Herring in 1905 who coined the term so unfortunately used in recent years—artificial hibernation—and stated that cold narcosis relieved pain like ether and chloroform narcosis, but that its use was not unattended by danger to life. In 1938 Temple Fay and Henry³ tried the effect of local cold on tumour growth, while in 1941 the term hypothermia was first used by Talbott.⁴

Bigelow and his colleagues showed that with progressive cooling the rectal temperature and oxygen consumption show an almost linear relationship. They also stressed the fundamental importance of controlling shivering, as even slight shivering doubles oxygen consumption. They finally pointed out that no oxygen debt was incurred by the tissues. The fact that hæmorrhagic shock was better tolerated by hypothermic dogs than by dogs at normal temperatures

respiration rate is reduced. There is a reduction in brain volume and cerebrospinal fluid pressure and in cerebral metabolism²⁶. The viscosity of the blood increases. The oxygen dissociation curve is shifted to the left and oxygen liberation to the tissues is hindered. The younger the patient the greater the safety, but the rectal temperature should not be allowed to fall below 26° C. Post operative pulmonary complications are increased while injury to local areas due to cold has been reported. At a temperature of 30° C (86° F) oxygen consumption is 70 per cent of normal and at 27° C it is 50 per cent of normal.

Some attention has recently been devoted to the study of the effects of hypothermia induced by surface cooling on the histopathology of vital organs (Knocker 1955)¹⁶. Changes are seen even when the experimental animals in this case dogs, are not cooled beyond the point generally regarded as ideal for surgery. The structural changes seen in the liver, kidneys and suprarenal glands are such as to make it questionable whether hypothermia by surface cooling ought to be used as a means of enabling patients to withstand circulatory arrest during operations on the heart. The cell changes resulting from hypothermia closely resemble those reported by other workers as resulting from various forms of stress and are perhaps due to tissue hypoxia. The unexplained deaths of animals during experimental hypothermia which have been reported may be due to these tissue changes becoming irreversible¹⁶. On the other hand the supporters of blood stream cooling have failed to produce these changes in their animals and suggest that the cold stress and the reflexes associated with surface cooling are the causes of the tissue changes and not the low temperature itself.

Methods of Producing Hypothermia

Cooling should be for as short a time as necessary for the successful completion of the proposed operation while rewarming should begin as soon as possible after the conclusion of the period when the heart is excluded from the circulation⁴⁰. There are, however, workers of experience who have seen bad results from too rapid rewarming³⁷. Techniques of hypothermia are not yet stabilized.

Surface Cooling This is the simplest method and is that used by Bigelow¹. The patient, after being anaesthetized is immersed in a cold bath surrounded by ice bags placed on a refrigerated mattress¹⁷ or wrapped in a refrigerated blanket²⁸. The time taken to reduce the temperature to 27-26° C varies from one to three hours, but the rate and extent of cooling are difficult to control. The so called

29° C¹⁵ The usual post operative oliguria does not occur under hypothermia while hypoxic damage to the liver is less serious than at normal temperatures. The myocardium is likely to go into a state of ventricular fibrillation at temperatures below 25° C and this fact not the effect on the brain limits the extent of hypothermia. This may be because during an operation the brain is at rest while the heart is still actively at work. The onset of fibrillation is influenced by the temperature direct handling of the heart hypoxia increase in the central venous pressure and disturbances in the intracellular electrolytic ratios especially of potassium and calcium.

Two new methods have been tried experimentally in an effort to provide a quiescent heart for intracardiac surgery. The first is to cool the heart locally to 7-10° C after the rest of the body has been cooled to 25° C and the inflow to the heart occluded. The second is the injection of neostigmine into the coronary circulation to produce a prolonged acetylcholine block which can be reversed at any time by atropine¹⁶. Another approach to the problem is that of Melrose and his co workers³¹ who have suggested the injection of potassium citrate into the coronary circulation of the hypothermic dog to produce a temporary circulatory standstill. The normal beat is restored after a period of asystole of fifteen minutes at 26° C by perfusion of the coronary circulation with fresh blood.

Clinical effects include a diminished oxygen requirement an absence of permanent damage due to enzymes which ordinarily accompanies periods of hypoxia a reduction in the work of the heart, a prolongation of the coagulation time of the blood and a reduction in the amount of anaesthetic required. A lowering of the pH of the blood is a feature of hypothermia and must be counteracted by hyperventilation. It is due to an increased amount of carbon dioxide dissolved in the plasma. On the other hand the addition of 5 to 10 per cent carbon dioxide to the inspired atmosphere during cooling unexpectedly reduces the incidence of arrhythmia and permits reversible cooling of experimental animals to 20° C³². There is interference with the renal and hepatic function which may retard excretion of anaesthetic agents heparin and glucose as the renal blood flow and glomerular filtration rate are reduced. Cardiac arrhythmia is seen in 70 per cent of patients who are cooled to 24° C or less and the ECG changes include increase in the contraction time prolongation of the P R interval widening of the QRS complex flattening and deep inversion of the T waves and depression of the ST segment. The blood pressure rises at first then falls gradually the pulse rate falls a direct effect on the pacemaker while the

this method in about forty five minutes and the blood pressure is sustained by a noradrenaline drip, as at temperatures below 28°C the cardiac output is so low that coronary flow might otherwise be inadequate. This also slows the heart rate. The patient is rewarmed to 35°C or until consciousness is regained before being returned to his bed. The danger of ventricular fibrillation is ever present and if it occurs, cooling is stopped while massage to restore tone, warming and electrical defibrillation of the myocardium are set in operation. If these measures do not restart a normal heart beat, the heart is again warmed with saline and intracardiac adrenaline or calcium chloride injected. No drugs are used to help cooling by this method but a search is being made for pharmacological agents which will make ventricular fibrillation less of a danger.

Thermometers used are either of the glass mercury type or else employ special electrical components. A useful heat measuring device has been described¹² which uses an electrical circuit working on the principle of measuring the difference between a known predetermined electric current and the varying current passed by a thermistor. By this means the temperatures between 20 and 40°C can be read on a scale discriminating to 0.1°C . The element in contact with the patient can either be a soft plastic tube or a needle for measuring intramuscular temperatures. A reading in the oesophagus gives a fair idea of the temperature of the heart, while one from the pharynx approximates to the temperature of the blood returning from the brain.

Re-warming

This can be achieved by immersion in warm water by wrapping in warm blankets or can be allowed to occur spontaneously. Burns must be avoided and the skin should not be raised above 40 – 45°C . Short wave diathermy can also be used the patient being placed in a high frequency field between two large electrodes. In perivascular cooling the blood is warmed directly.

Methods of Anaesthesia During Hypothermia

Destruction of drugs in the tissues and their elimination by the kidneys is interfered with by hypothermia and this necessitates the use of short acting and volatile drugs including that useful all purpose agent ether. Below 27°C , hypothermia itself causes unconsciousness. Small doses of intravenous barbiturate, gas oxygen and a relaxant to discourage shivering form a good anaesthetic combination. Chlorpromazine is the only drug with the dual

after cooling of 2 to 3° C takes place when surface cooling has stopped and is said to be due to the relief of the intense vasoconstriction which follows removal of the cold environment and the resulting perfusion of the cold surface tissues. This may cause severe inconvenience in cases in which the cooling has to be stopped suddenly. Surface cooling has been used with most success in infants and young children.

Body Cavity Cooling—In this method, chilled saline may be perfused into the pleural cavity after the thoracotomy,¹⁸ or cooling fluid may be circulated in a balloon placed in the stomach.¹⁹

Blood Stream Cooling (pervascular) This method obviates many of the complications such as reflex effects, tissue necrosis, etc. which may be seen after surface cooling and is used for intrathoracic operations. No after-cooling is seen once the procedure is stopped. It may involve carrying blood from an artery through a cooling coil and returning it to a vein,^{11, 7} it may also be carried out by passing blood from a vein through a cooling coil and returning it to another vein—veno-venous cooling. This latter method avoids injury to a major artery.^{20, 21} A refrigerating unit is placed outside the theatre while the blood cooling coil and pump are assembled near the patient. Simpler methods have been described^{25, 27} in which venous blood is made to circulate by a hand roller transfusion pump²⁶ through 16 feet of 4-mm Portex tubing coiled inside a 3 lb glass jar containing a mixture of solid carbon dioxide alcohol and saline. For rewarming this mixture is replaced by molten sodium thiosulphate which remains for a long period at a temperature of 46° C. Continuous ECG tracings are taken throughout the period of cooling and of operation and a glass or other thermometer placed in the pharynx or oesophagus records the temperature. After induction of anaesthesia the internal saphenous vein on the front of the thigh is cannulated and connected to the return end of a cooling coil while the other end of the coil leads to an ordinary saline infusion set. The decision to cool is delayed until the chest is opened and an accurate assessment for its need is made. If the technique is required then the afferent limb of the cooling coil is disconnected from the drip and pushed through a small opening in the right auricular appendage into the superior vena cava. The pump is set in motion and thus withdraws warm blood from the superior vena cava, cools it and returns it to the inferior vena cava via the internal saphenous vein. A recent improvement is the cannulation of both venae cavae via the right auricular appendage²⁷ instead of using the saphenous vein. A useful temperature is obtained by

injection of 50 per cent glucose solution, injection of neostigmine perhaps accompanied by the blocking by lignocaine of the sino auricular node and the junctional area and the inhalation of 5-10 per cent carbon dioxide. The induction of deliberate ventricular fibrillation to facilitate the surgeon's work has been described.³ On dogs defibrillation of the ventricles has followed the injection into the coronary artery of potassium chloride. This causes asystole which is then converted to normal contractile activity by massage and the intracoronary injection of calcium chloride.⁴ Rheumatic heart disease favours the occurrence of arrhythmias and may contraindicate hypothermia.⁵ Haemorrhage is more likely in cooled than in normal patients because of the increase in the blood coagulation time. Pulmonary oedema and atelectasis have also been encountered during hypothermia. Shivering must be prevented by the use of adequate anaesthesia together with muscle relaxants and some workers also employ chlorpromazine for this purpose. It appears that shivering is initiated from a centre in the thalamus and the autonomic response to cold is controlled from a centre in the posterior hypothalamus. Surface cooling may cause frost bite tissue necrosis and nerve injury effects not seen after blood stream cooling. Infection and also long term cerebral degeneration have been reported. Intravenous agents including anaesthetics and sedatives must be given in very small amounts as their excretion is greatly delayed at low temperatures.

The Uses of Induced Hypothermia

While the main use of this technique is to allow the heart and the brain to be safely deprived of oxygenated blood for periods up to ten minutes or a little longer so as to allow operations on the brain the heart and great vessels to be completed it has also been used in the treatment of various conditions in which it was considered that hypometabolism would benefit the patient. It may be that in the future hypothermia will be used exclusively in neurosurgery ischaemia being provided in the surgery of the heart by an artificial heart lung technique. At the time of writing (Sept. 1956) a mechanical pump oxygenator of the Gibbon type has already been successfully used in 40 human cases in which the heart has been excluded from the circulation for as long as 45 minutes. In the last fourteen operations in this series, only one patient died.⁶ The danger of spontaneous ventricular fibrillation precludes its use in general surgery except at such high temperatures that the beneficial effects are in doubt i.e. above 28° C. With increasing knowledge

action of inhibiting shivering and promoting vaso-dilatation²² and so may be used during the operation or in the premedication, but it is not obligatory. It does not prevent the release of ACTH by traumatic stress²⁴ and has little effect on cooling. It may even increase oxygen consumption. Glucose is metabolized more slowly in cold than in normal patients and so must be given by intravenous drip in reduced quantity. To combat respiratory acidosis which is likely to be present in cooled patients, the anaesthetist must hyperventilate the patient, except when the heart is isolated from the circulation then breathing is abolished.

In a method recently described²⁵ the patient is given chlorpromazine 50-100 mg by mouth the night before the operation, and one and a half hours before cooling is to commence, 50 mg of chlorpromazine is injected with hyalase intramuscularly. To this either pethidine or promethazine 50 mg may be added if the patient is anxious. One and a half hours later anaesthesia is induced with thiopentone 150-200 mg and d-tubocurarine chloride 30 mg. After intubation maintenance is by gas and oxygen. Now a cooled blanket or ice bags are applied to the patient, to his dorsal as well as to his ventral aspect and small additional doses of chlorpromazine are injected if vasoconstriction is pronounced. To prevent pooling of the blood in any one part of the body the position of the patient is changed every fifteen minutes.

Complications

Cardiac irregularities are seen with increasing frequency below a temperature of 33° C (91.4° F) and at 25° C ventricular fibrillation may be encountered. Many workers regard the safe lower limit as 30° to 28° C in adults and 28° to 25° C in children. Fibrillation is more common in old than in young subjects and is not always reversible by electrical defibrillatory techniques. It is most dangerous when it comes on before the cooling has reached the desired level. Continuous electrocardiographic monitoring is an essential part of the procedure. If the ventricles fibrillate cooling is terminated, the heart massaged to restore tone and the electric defibrillator applied. Should this grave complication make its appearance at a temperature low enough to allow the circulation to be interrupted or during the actual cardiectomy it is in the hands of some workers ignored as it prevents embolism and provides a quiet operating site. After the cardiac reconstruction is completed massage and electrical stimulation are employed to get spontaneous contractions going again. Other measures which may aid defibrillation include the intravenous

- 34 HOLZBAUER M and VOGT M 1954 *Brit J Pharmacol* 9 402
- 35 LUCAS B G II 1956 *Proc R Soc Med* 49 345 (June)
- 36 MARTIN P 1954 *Lancet* 2 1002
- 37 BROCK H 1956 *Proc R Soc Med* 49 347 (June)
- 38 ROSENTHAL H L 1956 *Proc R Soc Med* 49 358 (June)
- 39 BIGELOW W G CALLAGHAN J C and HOPPS J A 1950 *Ann Surg* 132 531
- 40 ROSS D N., 1956 *Proc R Soc Med* 49 365 (June)
- 41 MILLER, J A and MILLER F S 1954 *Surgery* 36 16
- 42 STRIDE, S D K. and DAVIS R W 1956 *Lancet* 2 309 (Aug 11)
- 43 GOLLAN F *et al* 1955 *Surgery* 38 363
- 44 KIRKALIN J W *et al*, 1956 *Ann Surg* 144 3 (July)

however, the onset of ventricular fibrillation may be less alarming and may come to be merely a cosmetic defect in the electrocardiogram⁴³

The tentative suggestion has been put forward by Gray²³ that by hypothermic anaesthesia we are preserving the adrenocortical responses to stress for the recovery period, and not depleting the reserve of the patient during the operation

Excellent recent articles on induced hypothermia are those by C F Scurr,³⁰ Lucas³¹ Brock³⁷ and Delorme³² Hypothermia is evolving rapidly and changes in outlook and techniques are occurring constantly

References

- 1 BIGELOW W G LINDSAY W K HARRISON R C and GORDON R A 1950 *Amer J Physiol* 160 125
- 2 SIMPSON S and HERRING P T 1905 *J Physiol* 32 305
- 3 FAY T and HENRY G C 1938 *Surg Gynec Obstet* 66 512
- 4 TALBOTT J H 1941, *New Engl J Med* 224 281
- 5 LABORIT H and HUGUENARD P 1951 *Pr méd* 59 1329
- 6 JUVENELLE A 1952 *Pr méd* 60 914
- 7 DELORME E J 1952 *Lancet* 2 914
- 8 LEWIS F J and TAUFIC M 1953 *Surgery* 33 52
- 9 SWAN H ZEAVIN I HOLMES J H and MONTGOMERY V 1953 *Ann Surg* 138 360
- 10 DUNDEE J W GRAY T C MESHAM P R and SCOTT W E B 1953 *Brit med J* 2 1237
- 11 BOEREMA I A et al 1951 *Arch Chir Neerl* 3 25
- 12 ROSS D N 1954 *Guy's Hosp Rep* 103 97 106
- 13 DELORME E J 1955 *Brit med Bull* 11 No 3 221 (Sept)
- 14 CHURCHILL DAVIDSON H C 1955 *Brit J Anaesth* 27 313 (June)
- 15 MILES H E and CHURCHILL DAVIDSON H C 1955 *Anesthesiology* 16 230 (March)
- 16 KNOCKER PHYLLIS 1955 *Lancet* 1 837 (Oct 22)
- 17 SCURR F C 1953 *Proc Roy Soc Med* 47 413
- 18 BLADES H and PIERPONT H C 1954 *Amer Surg* 140 557 (May)
- 19 KHALIL H H 1954 *Brit med J* 2 734
- 20 ROSS H N 1954 *Lancet* 1 1108
- 21 ROSS D N 1955 *Brit med Bull* 11 No 3 225 (Sept)
- 22 DUNDEE J W et al 1954 *Anaesthesia* 9 296 (Oct)
- 23 SENNING A 1952 *Acta chir scand* Suppl 171
- 24 ZEAVIN I VIRTUE R W SWAN H 1954 *Anesthesiology* 15 113 (March)
- 25 MURRAY D H and BRUCE D F 1955 *Lancet* 2 699
- 26 MIORNER G et al 1955 *Lancet* 2 593 (Sept 17)
- 27 SEDZIMER C B JACOBS D and DUNDEE J W 1955 *Brit J Anaesth* 27 93 (Feb)
- 28 INGLIS J M BIFFEN W M and D'ABREU A L 1954 *Lancet* 1 549
- 29 GRAY T C 1955 *Proc R Soc Med* 48 1083 (Dec)
- 30 SCURR C F 1955 *Proc R Soc Med* 48 1077 (Dec)
- 31 MELROSE G DREYER H BENTALL H H and BAKER J B E 1955 *Lancet* 2 21
- 32 DELORME E J 1956 *Anaesthesia* 11 221 (July)
- 33 LEWIS F J and NAZZI S A 1955 *Surgical Forum* Chicago

- 34 HOLZHAUER, M., and VOGT M 1954 *Brit J Pharmacol* 9 402
- 35 LUCAS, B G B., 1956 *Proc R Soc Med* 49 345 (June)
- 36 MARTIN F 1954 *Lancet* 2 1002.
- 37 BROCK R., 1956 *Proc R Soc Med* 49 347 (June)
- 38 ROSMOTT H L. 1956 *Proc R Soc Med* 49 358 (June)
- 39 BIGELOW W G CALLAGHAN J C and HOPPS J A 1950 *Ann Surg* 132, 531
- 40 ROSS D N., 1956 *Proc R Soc Med* 49 365 (June)
- 41 MILLER, J A and MILLER F S 1954 *Surgery* 36 16
- 42 STRIDE, S D A. and DAVIS R W 1956 *Lancet* 2, 307 (Aug 11)
- 43 GOLLAN F., et al 1955 *Surgery* 38 363
- 44 KIRKLYN J W et al 1956 *Ann Surg* 144 3 (July)

COLLAPSE, SHOCK AND RESUSCITATION

*Causes of sudden cardiac standstill—Types of cardiac standstill—
 Transthoracic and transabdominal cardiac pumping—Artificial
 circulation—Electrical defibrillation—Use of stimulant drugs—
 Prevention of cerebral œdema—Shock—Plasma Expanders—Blood
 Transfusion—Intra-arterial Transfusion*

CARDIAC RESUSCITATION

SOME real advances have been made in this subject within recent years, and sudden cardiac arrest in the operating theatre, and even in the ward and the out patient department, is no longer the certain fatality that it was not very long ago. It is now realized that sudden cessation of the heart beat may be due either to asystole (cardiac standstill) or to ventricular fibrillation, the former being the more common. This distinction is highly important as the treatment of the two conditions differs fundamentally, asystole requires cardiac pumping, misnamed cardiac massage, and ventricular fibrillation may demand electrical defibrillation in addition. The use of drugs in the treatment of cardiac standstill is also becoming better understood. In asystole, no observable movement of the myocardium takes place, but in ventricular fibrillation uncoordinated muscular contractions are present which from the functional point of view are totally useless as a means of expelling blood into the aorta.

The most common cause of cardiac arrest is hypoxia and this may result from too low an oxygen tension in the inspired gas mixture, respiratory obstruction due to one of its very numerous causes, underventilation, respiratory paralysis—central or peripheral, hæmorrhage or gross hypotension. A combination of two or more of these causes is to be especially feared, as for example a period of apnoea occurring in a patient with a very low blood pressure. Reflex cardiac arrest may be caused by certain drugs such as chloroform, ethyl chloride or cyclopropane, procaine amide, neostigmine, quinidine or adrenaline. It may also result from physical irritation of the heart during intrathoracic operations and during cardiac catheterization while a heart cooled to a temperature below about 25° C. may spontaneously develop ventricular fibrillation. Finally a heart subjected to gross pathological change such as coronary occlusion, pulmonary or air embolism may very soon cease to beat. *Reflex cardiac standstill from peripheral*

stimulation such as vagal inhibition is undoubtedly a cause, though a rare one, in clinical anaesthesia—and Johnstone¹⁵ gives fresh evidence for the view that cardiac arrest can be caused not only by vagal overaction but also by sympathetic overactivity and by a third mechanism that of hyperkalemia.¹⁶ In this carbon dioxide retention causes a rise in the plasma potassium content to cardiotoxic levels while a sudden fall in carbon dioxide in the plasma after a prolonged hypercarbia is followed by a still further increase in the plasma potassium with the possibility of the initiation of ventricular fibrillation. Johnstone makes a powerful plea for constant E.C.G. monitoring during major operations as only by this means can a timely warning of impending trouble be received by the anaesthetist.

Early diagnosis is imperative if the patient is to survive for if a period greater than four minutes intervenes between cardiac arrest and the commencement of an adequate artificial circulation irreversible brain changes are likely even if cardiac action is restored. The urgent need therefore is to institute and maintain a sufficient blood supply to the brain to prevent these irreversible changes. The myocardium itself is very resistant to periods of hypoxia an interesting contrast to its behaviour under hypothermia where it ceases to function at temperatures which leave the brain apparently unharmed.

The anaesthetist is sometimes faced with an apnoeic patient whose blood pressure is unrecordable whose precordium transmits no sound to the ear and whose peripheral arteries perhaps due to obesity are impalpable. There may come a time when he just does not know whether a patient is alive or dead. At this moment of uncertainty it is his immediate duty to inform the surgeon who may be in a position to palpate a large visceral artery. If all the evidence fails to confirm a beating heart massage must be commenced without further delay and is usually undertaken by a member of the surgical team. The anaesthetist meanwhile will lower the head of the table and see that efficient artificial respiration with oxygen or even if necessary with air by means of mouth to mouth breathing is maintained. He must not be tempted to waste time over a possibly difficult intubation as this can be performed later when the circulation has been restored. He will also try to get a needle into a vein so that blood and hypertensive drugs can be later administered. It is well however to remember that drugs designed to act on the myocardium will be useless if injected into a peripheral vein as they will not arrive at their site of action. Such drugs should be injected directly into the heart cavity or if the descending aorta is clamped,

into its lumen, so that they may be carried towards the openings of the coronary arteries

It is sometimes a difficult matter to decide whether cardiac massage should take place through the abdomen or through the chest, but in recent years opinion has come down greatly in favour of the latter route (McMillan, 1955)¹ If however an upper abdominal incision is already present, the aorta can be readily palpated and the heart itself felt through the diaphragm. If it is inactive it can be compressed between the hand and the chest wall—subdiaphragmatic massage—or by retracting the left lobe of the liver and incising the diaphragm behind the sternum² the heart can be compressed directly from within the pericardial cavity. If an effective heart beat results quickly from this method of resuscitation well and good but no more than thirty seconds should be spent on it and a transthoracic approach should be made forthwith. Compression of the heart from within the chest is the method of choice, as it not only enables a more efficient artificial circulation to be maintained and is less tiring but it is the only way, other than by electrocardiography, of differentiating between asystole and ventricular fibrillation. Without bothering about aseptic precautions, an incision is made in the left fourth or fifth intercostal space from a point near the sternal border, avoiding the internal mammary artery to the mid axillary line. The lung is retracted and the heart examined and frequent regular compression is at once started whatever the condition found. The ventricles must be squeezed empty and allowed to refill before the next compression the rate being about fifty or sixty each minute. Elevation of the legs and intravenous infusion may aid refilling. The operator's fingertips must not be used during the compression for fear of rupturing the flabby myocardium but the whole heart should be lifted up in the hand and compressed against the sternum.³

Once oxygenated blood has been squeezed out of the heart into the aorta and so continued for a short time, certain refinements and niceties of technique can be employed. A rib spreader such as Finochietto's can be put in place or one or more costal cartilages incised to allow the hands more room. The pericardial sac can be incised longitudinally and somebody to assist the surgeon in his truly exhausting work can be summoned. After further pumping the heart can be examined to see if it is in asystole or ventricular fibrillation. Asystole demands more compression until normal contractions are resumed. Should these return in a weak and inadequate form the myocardium should be stimulated by injection into the left auricle (or the thoracic aorta proximal to a clamp) of

3 to 5 ml of a mixture of noradrenaline in saline (1 ml of 1 1000 solution to 10 ml)² Raising the pressure of blood supplied to the coronary arteries and the brain can be helped by compressing the aorta just distal to the origin of the left subclavian artery⁴ if the transthoracic approach is being used or by occluding it in the region of the coeliac axis artery when abdominal massage is taking place. This artificial circulation should produce a systolic blood pressure of 40 to 60 mm of mercury⁵. If after some minutes of massage the normal heart beat does not appear, then drugs may be brought in as reinforcements and the first injection which should again be into the left auricle or into the aorta proximal to a clamp can well be 0.2 ml of 1 1000 adrenaline well diluted with saline or 3.5 ml of 1 10 000 noradrenaline. These drugs exert their action on the myocardium via the coronary arteries. A second drug which has proved useful is 10 per cent calcium chloride⁶ 3 to 4 ml of which should be similarly injected. This restores the tone of the heart muscle and may stimulate spontaneous contractions. Pumping is continued between injections which may have to be repeated but no case is to be regarded as hopeless until resuscitation has been continued for a long period. Drug injection may cause ventricular fibrillation by starting up multiple pacemakers. This demands the appropriate treatment by defibrillation.

The most effective way to abolish ventricular fibrillation is electrical defibrillation^{7, 8, 9, 10} which was suggested as long ago as 1899¹⁰. The aim is to pass an electric current through the heart strong enough to cause all its muscle fibres to have their refractory period simultaneously and then to initiate normal heart beats by massage and compression. After the circulation has been restored artificially two electrodes are applied to the heart so that a current passes between them. One shock may convert ventricular fibrillation to asystole or a rapid succession of shocks, each lasting 0.5 to one second may be required at increasing voltages up to 250 v. A period of compression is of course interposed between these attempts at electrical defibrillation. One example of a defibrillating apparatus is described by McMillan¹ and another (Stanley Cox Ltd—Stanco) gives a shock range from 100–350 volts of a duration of $\frac{1}{2}$ second. A large type of electrode should be used preferably covered with gauze moistened with saline, so that good contact with the myocardium is made to reduce the risk of burns to the heart. The operating team should wear rubber gloves to avoid electric shocks.

External electrodes have been successfully used without opening the chest to regularize a fibrillating heart¹¹ using electrical impulses

as an external cardiac pacemaker to maintain adequate cardiac contraction until spontaneous ventricular contractions reappear. For a successful result they must be applied within five minutes of the onset of fibrillation. The external defibrillator is similar in design to the direct defibrillator but the shock it delivers is much greater, the voltage used being 200-700 volts instead of the 120-250 volts which is usually adequate in the latter machine. Large copper electrodes covered with electrode paste are held by their insulated handles, firmly against the chest wall, one to the left of the sternum, the other on the anterior axillary line. Each shock lasts 15 seconds and is repeated at frequent intervals until a normal beat results. An electrocardiogram is necessary for the diagnosis of fibrillation to be made in the first place and also for the results of the shocks to be followed.¹⁷ This form of treatment is safe and it is easily applied but precious time must not be wasted, and if successful cardiac action is not soon seen the thorax must be opened without delay.

If electrical methods fail potassium should be available for trial in the form of 5 ml of 4 per cent potassium chloride. This has recently been successfully used to defibrillate the heart in induced hypothermia¹ but at a normal temperature is likely to be followed by difficulty in restarting the asystole which results from its injection even using calcium chloride and adrenaline. Intracardiac procaine 10 ml of 1 per cent solution has been advocated¹⁸ but it, like potassium, may make the resulting asystole difficult to reverse. Procaine amide has also been successfully used. Resuscitation may be unsuccessful in the presence of degenerative heart disease, hypoxia from any cause, low blood volume, toxæmia or brain trauma.

Once the circulation has been restored it may require augmentation and support by intravenous, intra-arterial or intra-aortic transfusion of blood in the case of hæmorrhage or of intravenous noradrenaline or other hypertensive drug. The chest should not be closed until spontaneous heart beats have been present for some minutes. Bleeding points must be secured, paying particular attention to the internal mammary artery while the pericardium should be sutured with interrupted stitches, not too tightly so that pericardial effusion can drain into the pleural cavity. The costal cartilages are then sutured if they have been divided and the chest closed using an underwater seal.

If effective artificial circulation is not established within three or four minutes of the cardiac arrest prophylactic treatment for possible cerebral œdema, the result of hypoxia, should be given. This may consist of the intravenous injection of 100 ml of triple

strength plasma—made from dried plasma—or of 50 per cent sucrose solution and such injections may be repeated at four hourly intervals. The intravenous injection of 0.1 per cent procaine hydrochloride solution once or twice daily has been recommended¹⁴ to relieve the vasoconstriction of the cerebral vessels associated with cerebral hypoxia.

For further details about this immensely important subject the reader is referred to articles by McMillan, Cockett and Styles³ and McMillan¹ and also to the book by R. M. Hosler, *Cardiac Resuscitation*, 1954 (C. C. Thomas Springfield U.S.).

SHOCK

This rather vague but highly important condition includes at least three pathological states. The first is not often seen by anaesthetists and is the reaction which sometimes results from emotion or other mild stress. It is characterized by bradycardia, a low blood pressure and a cold clammy skin. It is known as a vasovagal syncopal or fainting attack and treatment rarely presents any great difficulties.

Wound shock is hypovolemic shock and is usually due to acute haemorrhage. The patient becomes pale, cold and clammy, his pulse rate rises and his cardiac output is reduced. The treatment is immediate blood replacement or in its temporary absence plasma or plasma expander infusion. In the absence of treatment, the patient may be able to rehydrate himself spontaneously so that his cardiac output, blood pressure and venous pressure return again towards normal values. He will however be acutely anaemic and his blood as an oxygen carrier will be greatly handicapped so that his hypoxic myocardium may not be able to tolerate rapid transfusion. This is the so-called hyperkinetic phase of wound shock and should be treated by the slow transfusion of packed red cells. If on the other hand the patient is not able to rehydrate himself and remains untreated, peripheral circulatory failure follows and this may not respond to transfusion. This is the state of irreversible shock. Sympathomimetic agents have a place in the treatment of shock but their employment can be carried too far so that tissue hypoxia, increased capillary permeability and plasma leak has resulted from their continuous administration by intravenous drip. In small amounts however noradrenaline may redistribute available blood to better advantage.

Changes in the treatment of shock have not been numerous in recent years. While it was formerly the practice to warm up the

shocked patient this is now no longer so and some workers have deliberately cooled their shocked patients¹ A new approach to the treatment of the condition, at least from the point of view of the anaesthetist is its reversal by the intravenous injection of Ouabaine^{2,3,4} Ouabaine is strophanthin G, a digitalis like glycoside with a rapid action and a short duration of effect It should not be given to a patient who has recently been treated with digitalis or one of its congeners The intravenous dose is 0.5 mg² and its primary actions are to stimulate the myocardium and raise the peripheral resistance and hence the blood pressure It may be given either on its own or in addition to transfusion and may be specially useful in patients who have cold and blue extremities or tachycardia when the sympathomimetic amines might be contra indicated Horton and Armstrong Davison⁵ report good results in 84 per cent of their patients and strongly recommend its use in all patients with shock who do not improve after intravenous fluid has been given before, during or after operation They report no harmful results in their series of patients receiving this treatment

Plasma Expanders

These agents are a useful means of temporarily restoring the volume of circulating fluid provided the blood lost has not exceeded 20 per cent of the total blood volume Dextran (intradex, gentran, macrodex, plavolex, expandex) and polyvidone (periston, plasmosan, subtosan, polyvinyl pyrrolidone) are popular members of this group and experiments are taking place with gelatin Plasma expanders remain in the circulation longer than dextrose or saline because their molecules approach the size of the molecules in blood plasma and they are not filtered off by the kidneys In passing it should be emphasized that both dextrose and saline when given intravenously have an effect on the falling blood pressure which is not to be despised at such times as longer acting agents are not immediately to hand

Investigations have not yet been able to determine the ultimate fate of polyvidone (plasmosan) in the body Approximately 50 per cent is excreted through the kidneys within three days another 25 per cent is similarly excreted in the first ten days⁶ but the fate of the remaining 25 per cent is at present mysterious There is evidence that the material is deposited in the spleen liver bone marrow and lymphatic glands⁶

Reports of reactions to dextran come to hand from time to time^{7,8} and it would appear that this plasma expander can be antigenic to

man and can produce precipitins and also skin sensitivity. The degree of sensitivity as measured by skin tests seems to bear no relationship to the systemic reaction, while a second infusion after twenty one days does not enhance the sensitivity. Thus a look out should be kept for these reactions in all patients who receive dextran but as they are seldom serious they in no way preclude the use of this very useful agent. Reactions are more frequent in non anaesthetized than in anaesthetized subjects, and take the form of the usual allergic lesions such as urticaria, skin rashes, œdema and irritation of the mucous membrane of the upper respiratory tract. They are often ameliorated by the intravenous injection of antihistamine drugs.*

After intravenous injection of dextran containing radio active C 14, in the dog the urine shows 65 per cent to 70 per cent of the radioactivity with carbon dioxide in the expired air showing a further 4 per cent to 6 per cent. After an interval of 74 hours, 3 per cent to 5 per cent is present in the viscera. This leaves 25 per cent. of the dextran unaccounted for.¹⁰ The use of dextran is now much preferred to that of polyvidone.

Plasma is preferred to either dextran or polyvidone as a blood volume restorer in conditions where a hypofibrinogenæmia is likely to develop such as accidental hæmorrhage, a dead foetus in utero and hydatiform mole.¹¹

Blood Transfusion

Increasing experience is causing some workers to take a less serious view of incompatible blood transfusion than was formerly the case and Discombe¹ believes that most patients who receive incompatible blood recover spontaneously if their kidneys have not been damaged previously and if they have not been subjected to such biochemical insults as intravenous injections of, for example, sodium citrate sodium lactate or sodium sulphate. The treatment of this condition would now seem to be directed towards sparing the kidneys and so preventing death from uræmia which might result from anoxia of the renal tubules consequent on blockage of their capillaries by clumps of red cells. A litre of fluid should be given daily together with additional fluid equal to the volume, if any, passed as urine. Sodium chloride and sodium bicarbonate should be added to equal that lost and in addition dextrose and peanut oil should be given via an intragastric tube to provide calories. When diuresis follows, a careful watch must be kept on the electrolyte balance. In the presence of any oligæmic shock which results,

transfusion of compatible blood, or the intravenous infusion of noradrenaline or methyl amphetamine may be required

The best way to treat secondary anaemia in surgical patients is not by means of a blood transfusion just before or during an operation but by iron salts given by mouth during the days before admission to hospital. This can in many cases be supervised from an anæsthetic out patient clinic. It has been shown¹² that in blood stored in an acid citrate dextrose medium, the oxygen dissociation curve is shifted to the left while carbon dioxide transport is handicapped. Such changes are progressive with storage and the effects last several hours after transfusion. As a result of this shift the blood of a patient may for a few hours after transfusion be unable to release as much oxygen as it did before.

As a rough guide it would seem rational to use whole blood in cases of acute hæmorrhage in subacute hæmorrhage which has caused anaemia and in acute hæmolytic crises. Fresh blood which is always preferable to stored blood, is needed in cases of hæmophilia or of prothrombin deficiency. A transfusion of packed red cells, which should be given within twenty four hours of preparation to lessen the risk of bacterial growth consequent on the handling is often useful in patients with chronic anaemia unresponsive to other treatment before operation. It is also a useful means of treatment of the patient with aplastic anaemia. Small pool plasma serum or plasma expanders may be useful in patients with acute hæmorrhage before the arrival of blood or in patients who have suffered from trauma operative or otherwise. These agents are also useful in the treatment of burns. Fresh plasma like fresh blood may be indicated in hæmophilia and in hypoprothrombinæmia. Rapid transfusion of ice cold blood is harmful and ideally it should be heated to a temperature not exceeding 98° F.

Thrombophlebitis is a far too common sequel of intravenous therapy but its incidence may be reduced if certain types of rubber tubing are avoided in the apparatus or if plastic or latex tubing is used instead and if no drip is allowed to run for a period greater than eight hours. The addition of 0.75 to 1 ml of heparin (1:1000) to each 500 ml of solution reduces the number of sore arms and in this concentration the heparin is not sufficient to cause generalized bleeding even if the transfusion is run in during the actual operation. The addition of 0.01 per cent (10 mg per litre) of hydrocortisone to a drip will prevent this complication and may even cure existing thrombophlebitis.¹³

Intra-arterial Transfusions

This as a means of treatment of severe shock is not new, and was advocated by Crile fifty years ago. The usual technique involves the insertion of a cannula into the left radial artery about 2 in proximal to the radial styloid process. A solution of local analgesic is used for the skin incision; the artery is tied in continuity and a small incision made into the vessel proximal to the ligature. With experience it is no more difficult to cannulate an artery than a vein, while the risk of gangrene is slight. A source of pressure and a device for measuring it must be incorporated in the giving set, the idea being to force blood into the coronary vessels to stimulate the heart. Occasionally the femoral artery or the aorta may have to be used. Only blood must be used for intra-arterial transfusion. Some workers precede the transfusion with a stellate ganglion or a brachial plexus block to reduce arterial spasm, while for local spasm gauze soaked in 2 per cent procaine or 2.5 per cent papaverine may be used. The method is still under trial, but may be indicated¹⁴ in patients in severe shock who do not respond to intravenous transfusion, in cases of very severe hæmorrhage during operation, or when blood is in very short supply. This subject is further discussed in the section on arteriotomy.

It is now well known that circulatory collapse may be seen after relatively minor operations in patients who have recently been treated with cortisone. It appears to be a fact that the administration of this hormone leads to physiological inactivity of the patient's suprarenal cortex and this inactivity may be quite long lasting and may lead to an inadequate response to stress. It is thus wise to enquire from all patients before operation if they have been treated recently with cortisone or ACTH. If this is indeed so, then proper replacement dosage of the hormone must be prescribed in the days immediately before and after the surgical intervention. Even in patients who have not been recently receiving cortisone, good results have been claimed¹⁵ for the administration of blood followed by 100 mg of hydrocortisone in dextrose saline (see also Chapter 19).

References

Cardiac Resuscitation

1. McMILLAN I. K. R. 1955 *Brit med Bull* 11 No 3 229 (Sept.)
2. NICHOLSON J. C. 1942 *Brit med J* 1 385
3. McMILLAN I. K. R. COCKETT F. B. and STYLES P. 1952 *Thorax* 7 205
4. WIGGERS C. J. 1940 *Amer Heart J* 20 413

- 5 DRIPPS R D *et al* 1948 *Ann Surg* 127 592
- 6 KAY J H and BLALOCK A 1950 *Surg Gynec Obstet* 93 97
- 7 VETTEN K B WILSON V II CRAWSHAW G II and NICHOLSON J C
1955 *Brit J Anaesth* 27 2 (Jan)
- 8 HOOKER, D R *et al* 1933 *Amer J Physiol* 103 444
- 9 WIGGERS J 1936 *Amer J Physiol* 116 161
- 10 PREVOST J L and BATTIELLI F 1899 *J Physiol Path gén* 1 399
- 11 ZOLL, P M *et al* 1956 *New Engl J Med* 254 727
- 12 SWAN II *et al* 1953 *Ann Surg* 138 360
- 13 LEEDS S E 1953 *J Amer med Ass* 152 No 15 1409
- 14 COURVILLE C B 1955 *Anaesth & Analges* 34 74 (March-April)
- 15 JOHNSTONE M 1955 *Brit J Anaesth* 27 566 (Dec)
- 16 SEALY W C YOUNG W G and HARRIS J S 1954 *J Thorac Surg*
28 447
- 17 ZOLL P M *et al* 1955 *J Amer Med Assoc* 159 1428 Dec 10
- 18 MILSTEIN B B 1956 *Ann Royal Coll Surgeons Eng* 19 69

Shock

- 1 DELORME E J 1955 *Brit med Bull* 11 No 3 221 (Sept)
- 2 HORTON J A G and ARMSTRONG-DAVISON M H 1955 *Brit J Anaesth*
27 139 (March)
- 3 SANKEY B B and CRAWFORD T I 1950 *Anaesth & Analges* 29 148
- 4 WIGGERS C J 1947 *Amer Heart J* 33 633
- 5 WEESE H 1948 *Die Pharmazie* 3 337
- 6 STERN K 1952 *Proc Soc exp Biol N Y* 79 618 (April)
- 7 TARROW A B 1955 *Anesthesiology* 16 598 (July)
- 8 Report to National Research Council—U.S.A May 8 1952
- 9 TARROW A B and PULASKI E J 1953 *Anesthesiology* 14 359
- 10 PULASKI E J quoted by HARTMAN F W *et al* 1953 *J Amer med Ass*
152 1116 (July 18)
- 11 SCOTT J S 1955 *Brit med J* 2 290
- 12 DISCOMBE G 1954 *Lancet* 2 936
- 13 VALTIS D J and KENNEDY A C 1954 *Lancet* 1 119 (Jan 16)
- 14 BROWN A S *Lancet* 1953 2 745 (Oct 10)
- 15 LUNDY JOHN H 1955 *Proc Mayo Clin* 30 20 (Oct 5)
- 16 POLAK A 1956 *Lancet* April 21 p 484

CHAPTER 14

ANÆSTHESIA FOR DIAGNOSTIC PROCEDURES

*Bronchoscopy — Bronchography — Lumbar Encephalography—
Ventriculography — Cardiac Catheterisation — Angiocardi-
ography—Cerebral Angiography*

Bronchoscopy

THIS investigation is better done under topical analgesia in tolerant adult patients with large quantities of pus or blood in their air passages. The retention of their ability to cough is a safety factor in the post operative period. Topical analgesia is also needed even when general anæsthesia is used in order to dampen down the upper respiratory tract reflexes. There is an increased tendency to use general anæsthesia for bronchoscopy and this is due to the use of short acting muscle relaxants. Examination of the vocal cords can be done in the anæsthetized patient as the bronchoscope is being withdrawn, at a time when the patient's reflexes are returning to their normal active state.

One of the best methods of anæsthesia for bronchoscopy is that described by Macintosh¹. After the usual premedication, a Mitchell needle is placed in a vein and thiopentone and suxamethonium injected through it in doses averaging 300 mg and 80 mg respectively. After inflation of the lungs with oxygen the cords, pharynx, larynx and trachea and also the mucosa of the pyriform fossæ are carefully sprayed with 4 per cent cocaine solution after insertion of a laryngoscope, keeping the amount used in an adult to 200 mg or 5 ml of 4 per cent solution. In children the maximum dose of cocaine is 3 mg per kg of body weight. Care is taken to get the analgesic well down to the region of the carina. The lungs are again inflated until spontaneous breathing returns. If indeed it has not returned at the conclusion of the spraying and the surgeon is asked to pass the bronchoscope on the sleeping, breathing and quiescent patient. Additional thiopentone is injected if the patient requires it. This technique can be thoroughly recommended by the writers as a pleasant, safe and efficient one. It is also very useful for examination and biopsy of the vocal cords. A somewhat similar method described

by Churchill Davidson² requires the premedicated patient to be given an intravenous barbiturate and suxamethonium, followed by oxygen inflation and topical laryngeal analgesia. Additional relaxant is injected to keep the patient relaxed and apnœic and oxygen is gently forced down the bronchoscope periodically, via an 8 or 10 Magill tube cut short, which snugly fits the lumen of the bronchoscope. The artificial inflation is continued until the return of normal breathing and then the bronchoscope is withdrawn.

A method of anaesthesia which makes use of diffusion respiration has been described.³ After induction of anaesthesia with thiopentone and suxamethonium a portex tube with internal diameter of 3 mm is passed into the trachea so that its end lies near the carina and the body of the tube rests in the posterior commissure of the larynx. A flow of 3 litres of oxygen a minute is provided and bronchoscopy commences. During relatively short periods of apnœa the oxygen enables the patient to remain a good colour and for these short periods the hypercapnia need not be a serious hazard. Another variation of the same principle is the passage of a 16 F C.G. gum elastic endobronchial suction catheter reduced in length from 25 to 14 in with its distal end softened in hot water from the nose into the trachea. Through this runs a flow of 10 litres of N_2O and 5 litres of O_2 a minute. Bronchoscopy under thiopentone and suxamethonium then proceeds.⁴

An original method of making an apnœic patient breathe during bronchoscopy by means of a cuirass respirator has been described.⁵ It employs heavy premedication including pethidine 50–100 mg and chlorpromazine 25–50 mg, no topical analgesia and thiopentone (250–500 mg). The Monaghan type of cuirass is fitted, strapped and seal tested, a snug fit being necessary from the pelvis to the clavicles. Six sizes of plastic shell are available. Suxamethonium is injected and when apnœa results the respiratory pump is turned on. The cords and trachea are sprayed and additional barbiturate or relaxant injected as required. After the bronchoscopic examination in dry cases the patient is allowed to regain his tone and his reflexes, but in patients with much blood or pus in the air passages the bronchoscope is left in situ while aspiration is carried out until the reflexes are again active. The Monaghan respirator⁶ consists of a series of graduated sizes of plastic chest shells, a respiratory pump unit, a battery pump unit, and a hand pump. The pump when electrically controlled works at 10–15 cycles a minute and the relationship of inspiration to expiration is controllable. An air cushion of rubber makes a seal between the shell and the patient.

There are patients in whom a local analgesic might be desirable but who are not temperamentally fitted for it just as there are surgeons who get their best results in unconscious patients

Bronchography

Nearly every anesthetist who regularly helps in this diagnostic procedure has his own particular set of parlour tricks whereby he helps his radiological colleagues to get good pictures, while subjecting his patients to a minimum of discomfort and danger. In a method recently described for use in children¹ one minim of the ampoule of omnopon and scopolamine (gr $\frac{1}{4}$ and $\frac{1}{16}$) is given for each year of age and later anesthesia is induced with thiopentone (100-250 mg), or with nitrous oxide and oxygen if no veins are available. Suxamethonium 7 mg/stone is injected and later this can be repeated if necessary. The lungs are inflated with oxygen and a bronchoscope is passed suction is carried out and more oxygen is run into the side tube of the bronchoscope. After further inflation an endotracheal tube is passed to which is connected a Magill or a Cobb suction union and through the tube, gas oxygen and, if necessary, triene are inhaled until reflexes are controlled. After further oxygen ventilation the child is placed on the X ray table the contrast medium injected down the endotracheal tube and the pictures are taken after suitable positioning. Gravity aspiration and artificial ventilation soon result in a return of active breathing through a clear airway and the whole technique provides an absence of bronchospasm straining and hypoxia no psychic trauma and no risk of explosion. The radiological results are usually good.

Blind oral intubation in the conscious state has been used for bronchography in adults.² Premedication consists of atropine and two 250 mg capsules of methylpentynol. With the patient sitting up a number 9 Jaques catheter lubricated with 2 per cent lignocaine ointment is guided into the larynx while the patient pulls out his tongue with his chin forwards and pointing slightly upwards. Coughing and straining soon cease and the contrast medium is then injected down the tube and after the patient is positioned, pictures are taken. Another method utilizing indirect laryngoscopy³ is a modification of that described in the United States by Gianturco. The patient sits facing the operator who applies topical analgesia to the larynx and visualizes the superior laryngeal aperture in his laryngeal mirror. The blade of the special instrument is now passed over the base of the tongue and by its means a catheter is guided into the larynx and strapped to the side of the face.

Anæsthesia for Radiological Investigations in Neurosurgery

General anæsthesia may be required in children or in nervous or uncooperative adults

For lumbar encephalography¹⁰ induction may be by thiopentone and gallamine 60 mg (1.5 ml) of the latter made up to 10 ml with 5 per cent thiopentone with 2 ml of this mixture per stone being used. The larynx is exposed, sprayed with an analgesic solution and a non kinking flexo metallic tube is inserted into the trachea. If this is difficult, a nasotracheal tube is passed. Acute flexion of the neck will be necessary during the examination so kinking of the airway must be prevented. In small children it may be necessary to induce anæsthesia outside the X ray room with a volatile anæsthetic. The patient is then sat up and suitably fixed, and nitrous oxide, oxygen, trilene are given. A lumbar puncture is performed and 30–40 ml of air are injected in divided amounts. The radiologist will want to move the patient and this must be provided for. Light anæsthesia with absence of reflex response is aimed at. On return to the ward, signs of raised intracranial pressure must be looked for, such as prolonged unconsciousness, and ventricular tapping may become necessary.

For ventriculography, air is injected into the ventricles in the operating theatre and the patient then taken to the X ray department. He is kept either prone or supine throughout the examination.

Cerebral angiography which is not done under local analgesia requires a light level of anæsthesia with absence of coughing. A suitable technique¹⁰ includes thiopentone, gallamine, analgesic spray, endotracheal intubation and N₂O/O and trilene anæsthesia, the breathing tube being led over the patient's chest. When the patient has settled down the tissues round the common carotid and vertebral arteries are infiltrated with local analgesic solution to which has been added 1 ml of 5 per cent papaverine¹¹ and after a short pause a needle is inserted into the artery and 8–10 ml of 35 per cent uriodone injected. After minor fluctuations in blood pressure either an immediate fall of anything between 20 and 100 mm Hg or a more gradual and less severe fall in pressure is seen.¹¹ The first type may last minutes or hours—occasionally pressor drugs are necessary for its reversal. It is most often seen in patients with recently ruptured aneurysms or in those who have sustained injury to the pre optic portion of the hypothalamus or basal ganglia. The second type is perhaps due to cerebral œdema from damage by the contrast medium to the vascular endothelium. In a recent series of 350 examinations¹¹ no serious complications have arisen.

Anæsthesia for Cardiac Catheterization and Angiocardiography

Cardiac catheterization is a widely used diagnostic procedure to select patients for the surgical repair of acquired and congenital heart disease. The test involves the insertion of a ureteric type catheter up a peripheral vein into the right auricle and from there into the superior or inferior vena cava and across defects between the cardiac chambers and the great vessels. Gas analysis and pressure tracings can be taken via the catheter and from a needle inserted into an artery. The addition of volatile anæsthetics or anæsthetic gases may invalidate the tests, while emotion, reflex movements or other abnormal states due to the anæsthetic are also likely to be undesirable. The investigation is a major risk but is not a painful one, so that adults and older children can be controlled by sedation. Younger children usually require light general anæsthesia. The investigation is carried out in the X ray room and may take several hours. The room will be dark for most of the time. The patient is often in poor condition, cyanosed perhaps with large quantities of sputum. Reflex bronchospasm and coughing must be prevented by the anæsthetic. So that the anæsthetist can keep in touch with the patient's cardiac action Inglis has described¹² a telephonic stethoscope which amplifies the heart sounds.

Various methods of anæsthesia have been described. Under local analgesia in older children unpleasant symptoms such as pain in the arm vertigo vomiting coughing respiratory arrest of short duration hypotension shock and even death have been reported. Basal narcosis forms part of several anæsthetic techniques^{10 13 14} some workers preferring rectal thiopentone¹³ others bromethol¹¹. When the patient is placed on the X ray table intravenous thiopentone is given intermittently and is supplemented with 10-mg doses of pethidine until the respiration rate approaches that of normal sleep¹². If there is no vein gas and oxygen are used for induction and when the catheter is in place it is used for intermittent thiopentone injections¹⁴. The passage of an endotracheal tube is seldom necessary but all facilities for urgent intubation in an emergency must be available. The catheter is inserted after thorough infiltration of the vein and its surrounding tissues with local analgesic solution to prevent venous spasm. Some workers use a heparin saline drip to keep the catheter patent. The anæsthetist should use special gloves to protect himself against damage from X rays.

For angiocardiography when no blood sample is taken for analysis gas oxygen and trilene can be given in addition to the basal narcotic and thiopentone intravenously.

References

- 1 MACINTOSH R R 1954 *Anæsthesia* 9 77 (April)
- 2 CHURCHILL DAVIDSON H L 1953 *Anæsthesia* 8 128 (April)
- 3 CHEATLE, C A and CHAMBERS K B 1955 *Anæsthesia* 10 171 (April)
- 4 BUCK H A 1955 *Anæsthesia* 10 313 (July)
- 5 GREEN R B 1955 *Anæsthesia* 10 369 (Oct)
- 6 KILLEITER W H WILSON E A B RUSSELL, R. W R. and STOTT F D.,
1952 *Brit med J* 2 413
- 7 LOVE S H S and MORROW W F K. 1954 *Anæsthesia* 9 74 (April)
- 8 BARKER J F 1955 *Brit med J* 1 1031 (April 23)
- 9 SALISBURY B 1955 *Lancet* 2 650 (Sept 24)
- 10 COLES P F DE C 1953 *Anæsthesia* 8 186 (July)
- 11 BROWN A S 1955 *Anæsthesia* 10 346 (Oct)
- 12 INGLIS J M 1954 *Anæsthesia* 9 25 (Jan)
- 13 COPE, D H P 1953 *Brit J Anæsth* 25 212 (July)
- 14 GIPSON J B 1953 *Anæsthesia* 8 269 (Oct)

CHAPTER 15

ANÆSTHESIA FOR THORACIC SURGERY

Methods of Controlling Secretions—The Carlen's Double Lumen Catheter—The Vellacott Endobronchial Tube—The Stueritzbecher Tube—The Macintosh and Leatherdale Tubes—Repair of Oesophageal Atresia in the Newborn

DURING the decade beginning in 1946, anaesthesia for intrathoracic operations became relatively stabilized and now most workers induce anaesthesia with an intravenous barbiturate, inflate the patient with an oxygen and nitrous oxide mixture, intubate with the aid of a relaxant assist or more usually control the respiration while the chest is open and maintain analgesia with small amounts of pethidine. Cyclopropane is used less frequently than it was as apnoea can now very readily be produced by the myoneural blocking drugs and non explosive agents are preferred. Similarly, the use of ether is not very common in Britain although it still has its warm advocates who give good reasons for its continuance especially for induction in order to relax bronchial muscle and show excellent results from its use¹. When the chest is open respiration must be either assisted or more usually controlled. In assisted respiration, the respiratory centre and muscles retain some activity and pressure on the reservoir bag is synchronized with inspiration while it is completely withdrawn to allow full passive expiration to take place. In controlled breathing either the respiratory centre or the respiratory muscles or both are inactive and artificial intermittent positive-pressure breathing is taken over by the anaesthetist who can control its rate and depth. Full periodic inflation of the lung being operated on is not necessary, but complete reinflation at the end of the procedure is important. The whole lung should be carefully examined to see that it expands and if necessary it should be gently massaged. With the patient in the lateral position and one side of the chest open an elevating mechanism should be avoided to prevent the lower lung being compressed between the elevating pad and the mediastinum. The control of secretions and the prevention of transbronchial spread during operation remain a problem although the number of patients with copious secretion who come to operation is very much smaller than was the case before the introduction of the newer chemotherapeutic and antibiotic drugs.

Transbronchial spread of secretion during lung resection² has in the past been prevented by endobronchial intubation usually of the left main bronchus using an inflatable rubber cuff,^{3,4} by the use of bronchial blockers^{3,4,5} by gauze packing of the affected bronchus through a bronchoscope,⁶ and by the adoption of the very steep tilt with the patient in the lateral position⁷ or with the patient lying prone⁸ so that secretions are carried into the trachea where they can be removed by suction rather than into the other parts of the lung. The prone position is not acceptable to all surgeons however and is not advisable for resection of the middle lobe, lingula or an anterior segment. Moreover the lateral position has advantages from the anæsthetist's point of view as during a thoracotomy the function of the lower lung is increased partly due to the gravity shunt of blood into the lower lung.⁹

Much effort and ingenuity have been and continue to be employed in overcoming these difficulties. One result is the use of the Carlens

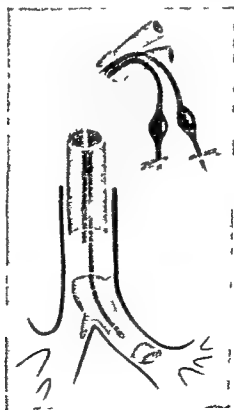


FIG 60 Carlens double lumen intrabronchial tube
(Björk, Carlens and Friberg, *Anesthesiology* 1953)

double lumen catheter^{10 11} which was originally used for differential bronchspirometry in 1949,¹² but has since then been employed for differential endobronchial anaesthesia during operations on the lungs. This double lumen catheter is made of rubber and is moderately rigid. It is obtainable in two or three sizes, the lumen of each side being 7 or 6 mm in diameter and the outside measurement is 13 or 11 mm according to size. It is provided with two inflatable cuffs and has two built in curves. A small solid rubber hook is placed just above the lower curve and ensures correct positioning of the tube by engaging the carina. The portion of the catheter below the lower curve consists of the channel for the left bronchus and is about 4 cm in length. On it is an inflatable cuff so constructed that the left main bronchus can be made airtight without obstruction of the left upper lobe bronchus. Just above the carinal hook there is a lateral opening which corresponds to the lumen of the right bronchus and above this opening is the second inflatable cuff on the endotracheal portion of the tube. The small inflating tubes leading to the balloons have pilot bags at their proximal ends. In addition to isolating a healthy lung to prevent spread of disease, the use of the Carlens tube enables the anaesthetist to aspirate pus or blood from each lung without interrupting the anaesthesia, and to inflate or deflate, temporarily or permanently, either lung.

This tube requires neither fluoroscopy nor bronchoscopy for its insertion, but is accurately placed in the unconscious patient through the ordinary laryngoscope. It is also possible to intubate the conscious patient under topical analgesia,¹¹ in which case the patient sits facing the anaesthetist who uses a headlight and indirect laryngeal mirror and introduces the tube between the cords over a curved metal stylet. Some workers tie the rubber hook to the plane of the tube with a moistened silk thread until with a slight rotating movement, the tube is passed through the larynx after which the slip knot is untied. Other anaesthetists omit this. The catheter is gently pushed downwards and turns automatically to the left when the stylet is withdrawn. The tip now enters the left main bronchus and the hook engages the carina. Once in position the cuffs are inflated and their friction with the mucosa together with the carinal hook keep the tube in position. During left pneumonectomies closure of the left bronchus is carried out after deflating the balloons and withdrawing the tube for 5 cm. Left endobronchial suction is used as a preliminary measure to remove secretions. Afterwards the proximal cuff also is reinflated and the apparatus used as a single lumen endotracheal tube. When the left main bronchus is obstructed, this tube cannot be used.

Endobronchial anæsthesia may be specially useful in cases of tuberculosis with cavitation or secondary suppuration behind a bronchial stenosis, in chronic lung abscess or in secondary suppuration behind a bronchogenic neoplasm. In cases of sudden hæmorrhage the method may be most helpful.

The special advantages of this method of endobronchial anæsthesia are that once the chest is open, the diseased lung can be deflated to provide greater space for operating; either lung can be inflated at will, in cases requiring frequent aspiration of secretions, one lung is always ventilated while the same plane of anæsthesia can be easily maintained. Carlens and his associates¹¹ are of the opinion that free passage and good ventilation to the healthy lung during the entire operation is provided more easily by endobronchial than by endotracheal anæsthesia. They use a mechanical respirator, inspiration being produced in the relaxed patient by increasing the pressure to 15-20 cm. of water then suddenly dropping it to zero allowing passive expiration to take place. The expiratory phase is of course longer than the inspiratory phase. When one lung has been clamped

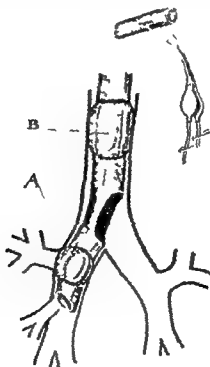


FIG. 61 The Vellacott endobronchial tube (*Brit J Anæsth.*)

off, the inflationary pressure may have to be increased to 25 cm of water because of the increased resistance, the result of the single lumen. In several thousand cases in which the Carlens tube has been used it has been shown that the venous return and the minute volume are not diminished enough to decrease the blood pressure while blood gas analyses have repeatedly proved the efficiency of the method.¹¹ Considerable obstruction to respiration is however experienced when spontaneous respiration is allowed to persist.¹²

Vellacott has described an endobronchial tube¹³ for use in cases of right upper lobectomy with much sputum, in those patients in whom it is impossible to place a short cuffed Magill blocker in the right upper lobe bronchus. It is also recommended for the occasional case of bronchopleural fistula and empyæma which follows a right upper lobectomy. When such a fistula has to be closed it may be desirable to block off what is left of the right upper lobe bronchus to prevent entry of pus and blood into the bronchial tree which may collect while the surgeon is getting down to the fistula. An ordinary blocker placed in the right main bronchus occludes the right upper lobe but in addition inactivates the right lung, while the same disadvantage is seen if an endobronchial cuffed tube is placed in the left main bronchus. The tube illustrated is a single lumen latex tube reinforced by a spiral wire and stiffened with a malleable stylet built into the wall of the tube. It is provided with two inflatable cuffs, one (A) being placed opposite the right upper lobe bronchus and occluding it the second (B) remaining in the trachea. Between the two cuffs is a deeply cut gap 1 inch long in the wall of the tube, which allows aeration of the left lung through its main bronchus. A similar reinforced latex tube has been designed for the left main bronchus for use during right pneumonectomy but employs only the distal cuff and has no lateral gap. These tubes must of course be inserted over a bronchoscope.

The use of the combined endotracheal tube and bronchus blocker of Stuertzebecher¹⁵ has been described by Oech.¹⁶ This tube is manufactured in Western Germany in two lengths and a number of sizes and the endobronchial blocker projects distal to the opening of the tube. For pneumonectomies in which only the main stem bronchus need be occluded, the blocker extends 7 cm beyond the end of the endotracheal tube. When lobectomy or segmental resection is planned the blocker is longer by 2 cm for deeper endobronchial occlusion. There are two inflatable cuffs one on the blocker with a rough outer surface to prevent displacement and the other on the endotracheal part. Each of these has an inflation tube

and pilot balloon, while a third small tube allows for aspiration from the end of the blocker which takes the form of an olive shaped metal tip for X ray localization. Before insertion through the laryngoscope a curved wire stylet is threaded into the blocker tube to give stiffness and to enable each main bronchus to be entered blindly. After the bronchus has been entered, the stylet is withdrawn and the cuff of the blocker inflated. Proper positioning can be checked by radiography or by auscultation over the chest. The use of this piece

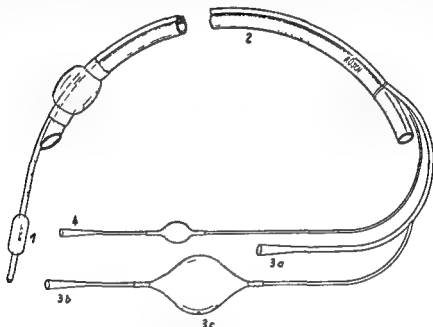


FIG 62 The Stuertzebecher combined endotracheal and bronchus blocker

of apparatus allows either the entire lung or the lobe to be isolated from normal tissue. Secretion can be aspirated without interfering with ventilation in the remaining part. The blocked off parts can be expanded or collapsed thus facilitating

Two carefully designed and manufactured endobronchial blocker and endotracheal tube by Stuertzebecher and one endobronchial blocker by Macintosh and Leatherdale¹⁷. Their advantages are: Once in position the endobronchial blocker is not easily dislodged, it lies closely to the line of the trachea and

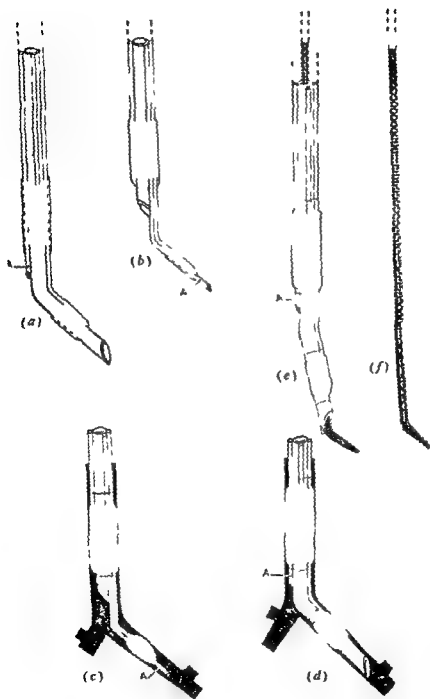


FIG 63 (a)-(f) Combined endobronchial blocker and endobronchial tube (Macintosh and Leatherdale *Brit J Anaesth*)

and pilot balloon while a third small tube allows for aspiration from the end of the blocker which takes the form of an olive shaped metal tip for X ray localization. Before insertion through the laryngoscope a curved wire stylet is threaded into the blocker tube to give stiffness and to enable each main bronchus to be entered blindly. After the bronchus has been entered, the stylet is withdrawn and the cuff of the blocker inflated. Proper positioning can be checked by radiography or by auscultation over the chest. The use of this piece

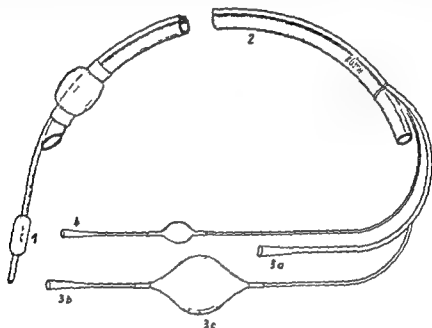


FIG 62 The Stüertzbecher combined endotracheal tube and bronchus blocker

of apparatus allows either the entire lung or the lobes to be resected to be isolated from normal tissue. Secretion can be aspirated without interfering with ventilation in the remaining pulmonary tissue. By aspiration or inflation the blocked off parts of the lung can be either expanded or collapsed thus facilitating identification and dissection.

Two carefully designed and manufactured tubes, a combined endobronchial blocker and endotracheal tube, a development of the Stüertzbecher tube, and one endobronchial tube have been described by Macintosh and Leatherdale.¹⁷ They are said to possess three advantages. Once in position the endobronchial tube or endobronchial blocker is not easily dislodged as their shape conforms closely to the line of the trachea and the left main bronchus. Both

oxygen is insufflated into the lungs, and a size 00 armoured endotracheal tube is gently placed in the trachea. A mixture of equal parts of oxygen and nitrous oxide is given by controlled respiration using a small to and fro absorber with a controllable leak and a total gas flow of 3 or 4 litre/min or other suitable circuit which prevents accumulation of carbon dioxide. It helps the surgeon if a fine rubber catheter is passed from the nose into the upper end of the œsophagus to help identify it. The transpleural approach is the one usually employed by the surgeon. When the operation is completed, neostigmine is injected if it is indicated using 0.03 mg/lb with atropine gr $\frac{1}{16}$. As early as possible during the operation the surgeon should be asked to close the tracheo-œsophageal fistula so that anæsthetic gases do not pass into the alimentary tract causing distension. Blood loss is replaced as necessary but should seldom exceed 100 ml. It is well to remember that in these small babies the tidal volume may be as little as 20–25 ml. There are some workers of experience who use no anæsthetic agent whatever but rely solely on a relaxant to produce apnoea and oxygen inflation through an endotracheal tube. Their view is that the newborn child does not really appreciate pain and so can be spared the injection or inhalation of all potentially toxic agents. Open ether induction, followed by endotracheal ether oxygen by controlled respiration is another safe technique.

References

- 1 BEECHER H K 1952 *The Principles Problems and Practices of Anæsthesia for Thoracic Surgery* p 43 et seq Springfield U.S. C C Thomas
- 2 GALE J W WATERS R M 1931 *J thorac Surg* 1 432
- 3 MAGILL I W 1935 *Proc R Soc Med* 29 649
- 4 ARCHIBALD E 1935 *J thorac Surg* 4 335
- 5 RUSBY L N and THOMPSON V C 1943 *Post grad med J* 19 43
- 6 CRAFOORD C 1938 *Acta chir Scand Suppl* 54 65
- 7 BEECHER H K 1940 *J thorac Surg* 9 202
- 8 OVERHOLT R H and LANGER L 1949 *The Technique of Pulmonary Resection* p 17 Springfield U.S. C C Thomas
- 9 ROTHSTEIN E LANDIS F H and NARODICK B G 1950 *J thorac Surg* 19 821
- 10 BJORK V O and CARLÉN E 1950 *J thorac Surg* 20 151
- 11 BJORK V O CARLÉN E and FRIBERG O 1953 *Anesthesiology* 14 60
- 12 CARLÉN E 1949 *J thorac Surg* 18 742
- 13 MACKRELL T H et al 1954 *Anesthesiology* 15 209 (March)
- 14 VELLACOTT W H 1954 *Brit J Anæsth* 26 442 (Nov)
- 15 STÜERTZBECHER F 1953 *Der Anæsthesist* 2 151
- 16 OECH E R 1955 *Anesthesiology* 16 468 (May)
- 17 MACINTOSH R R and LEATHERDALE R A L 1955 *Brit J Anæsth* 27 556 (Nov)
- 18 PAYNE J P 1955 *Brit J Anæsth* 27 388 (Aug)
- 19 *Brit J Anaesth* 1956 28 225
- 20 MANSFIELD R E 1956 *Proc World Congress of Anæsthesiologists (1955)* p 55 Minneapolis Burgess

endobronchial tube and blocker can be passed blindly without the aid of a bronchoscope while a small tube (A in Fig 63 (a)-(e)) is incorporated which can be used either to aspirate secretions from the non functioning lung or to distend it to help the surgeon's dissection.

When the left lung is to be operated on, the cuffed tube and its incorporated blocker is used. The blocker is angulated so that it enters the left main bronchus. When the endobronchial cuff is inflated the left lung is isolated (Fig 63 (c)) so that when the reservoir bag is compressed only the right lung expands. The blocker tube can be used for suction or to supply oxygen to the left lung.

If the right lung is to be the site of the surgeon's attention the endobronchial tube is passed into the left main bronchus (Fig 63 (d)) under an intravenous barbiturate topical analgesia and a muscle relaxant. First through a laryngoscope it is inserted into the trachea and is then advanced blindly when because of its shape it will tend to enter the left bronchus coming to a stop when the angle of the tube is halted at the carina. The distal end of the tube now lies just short of the upper lobe bronchus. The endobronchial cuff is now inflated and the cuff surmounting the endotracheal portion too if required. Through a small tube (A Fig. 63(d)) oxygen can be supplied to the right lung or secretions aspirated from it. Should blind endobronchial intubation prove difficult the tube should be withdrawn and eased over a firm rubber director the tip of which is guided into the left bronchus. After the tube has been pushed down into position the director can be withdrawn (Fig. 63 (f)).

Blind methods of endobronchial anaesthesia are very attractive but it has been pointed out by members of the anaesthetic staff of the Brompton Hospital for Diseases of the Chest in London that blind bronchus-blocking should not displace direct vision placement of blockers partly because in expert hands the latter method is more accurate.¹⁸ The word expert in this connection is very important.

Repair of Oesophageal Atresia in the Newborn

This operation was first successfully recorded in 1943 by Haight and Townley in the United States and the anaesthetic management calls for a delicate technique and careful handling.¹⁸ Before the anaesthetic is begun a fine polythene tube should be inserted into the internal saphenous vein and through it a drip of fifth normal saline and 4.3 per cent. dextrose established. Premedication is atropine gr $\frac{1}{32}$ and for induction thiopentone 2.5 to 5 mg/lb of body weight and d-tubocurarine 1 mg per 5 lb of body weight are given into the drip. The larynx is aspirated free of secretions

year.⁶ The operation of mitral valvotomy as it is performed to day dates from the report by Bailey of Philadelphia in 1949.⁷

The opening through the mitral valve in normal people measures 4 sq cm and great quantities of blood can flow through it with only a small increase in left auricular pressure. If through endocarditis and fibrosis the size has constricted down to 1 sq cm, the patient is delicately balanced on the brink of disaster and very slight changes may upset his equilibrium with the production of hæmoptysis or acute pulmonary œdema. Such patients have little cardiac reserve and little increase in cardiac output is possible. Mitral stenosis delays filling of the left ventricle and although filling continues during diastole if the cardiac rate is increased, diastole is shortened and less time for filling is allowed and stroke volume is decreased. Thus tachycardia must be prevented.

The pre operative preparation is in the hands of the cardiologist and usually includes attention to the emotional as well as to the physical needs of the patient. Electrolyte balance, digitalization, dental sepsis, anaemia and nutrition are likely to be dealt with, breathing exercises are taught and minor ills remedied if possible. Blood must be prepared for transfusion in case it should be needed.

Premedication is very important as adequate sedation in severe cases is a factor in the prevention of pulmonary œdema. Secretions from the upper respiratory tract must be suppressed and reflex tone must be reduced. Many workers employ atropine but others fear its use as it may cause tachycardia.⁸ Pentobarbitone in 100 mg doses by mouth with morphine 10 mg (gr $\frac{1}{2}$) hypodermically, form a good combination while omnopon and scopolamine⁹ and pethidine scopolamine have been favoured by others.¹⁰

The induction of anaesthesia takes place in that position which is most comfortable for the patient whether it be lying or semi sitting. The whole procedure must be perfectly smooth and completely devoid of coughing and straining. A stormy induction may readily precipitate an attack of pulmonary œdema. Many workers⁹ commence by allowing the patient to inhale pure oxygen for several minutes and in severe cases topical laryngeal analgesia should be applied before consciousness is lost so that the chance of laryngeal spasm and cough is lessened. To produce unconsciousness thio pentone can be given in 2.5 per cent solution (up to 150-500 mg) and this can be followed by a relaxant such as d tubocurarine 15-30 mg followed by inflation with oxygen. Some workers prefer gallamine to d tubocurarine because of its shorter action and more complete reversal by neostigmine¹⁰ and are prepared to overlook

CHAPTER 16

ANÆSTHESIA FOR OPERATIONS ON THE HEART AND GREAT VESSELS

Classification of Congenital Heart Disease—The Conduct of Anæsthesia for Cardiac Operations

THE following types of congenital heart disease have been described some of which may be suitable for operation¹ Cyanotic cases include Fallot's tetralogy, when treatment may be an indirect operation such as Blalock's systemic pulmonary anastomosis or Pott's aorto pulmonary anastomosis or by the direct operation of pulmonary valvotomy Other types of disease in this group are simple pulmonary stenosis with right to left intra atrial shunt (pulmonary valvotomy), pulmonary atresia tricuspid atresia (subclavian pulmonary anastomosis) dextrocardia and isolated laevo cardia and other complex abnormalities Acyanotic cases include six different conditions, three with left to right shunts and three with various degrees of obstruction to blood flow The former group includes atrial septal defect (the surgical management being closure), ventricular septal defect (closure) and patent ductus (closure) The latter group includes simple pulmonary stenosis (valvotomy), aortic stenosis (valvotomy) and coarctation of the aorta (resection and anastomosis or grafting)

Acquired heart disease which may now or in the near future be suitable for surgical treatment includes² wounds of the heart acute pulmonary embolism (removal of clot) Pick's disease (pericardiectomy) ischæmic heart disease (various experimental operations designed to direct a blood supply from the chest wall to the myocardium) mitral stenosis (mitral valvotomy) mitral incompetence (experimental operations) aortic stenosis (aortic valvotomy) aortic incompetence (insertion of a plastic valve into the descending aorta)³ and tricuspid stenosis (tricuspid valvotomy)

As mitral valvotomy is the commonest operation performed on the heart to day the management of anæsthesia for this operation will be described, but it does not differ greatly from that employed for other cardiac procedures The modern attack on the diseased heart commenced when Rehn of Frankfort was successful in suturing a wound of the myocardium in 1897⁴ Souttar at the London Hospital successfully cured a case of mitral stenosis in 1925⁵ and a similar case by Pribram was reported from Germany in the following

It is sometimes a problem to know when to give additional relaxant or analgesic drugs but the signs do not differ in these cardiac operations from those seen in other thoracic or abdominal operations. More relaxant may be needed if the resistance to inflation becomes greater if spontaneous respiration shows signs of returning or if lung formerly out of sight, trespasses into the wound. More analgesia may be required if the patient shows movement. thiopentone for coarse movement, pethidine for fine muscular activity, as in the face finger or pharynx. The risk of displacement of emboli from the left auricle or auricular appendage can be reduced if the anaesthetist compresses the carotid arteries for a short while during critical periods of the operation as when the appendage is first palpated or when the valvotomy is being performed. Occasionally torrential hæmorrhage will temporarily defeat the surgeon and at such times large volumes of blood must be rapidly transfused.

The control of arrhythmias¹⁶ is an important part of the management of cardiac operations and constant ECG monitoring is helpful. Irregularities of the heart may be due to the state of anaesthesia to vagal stimuli from the endotracheal tube to rib spreading and to manipulation of the heart¹⁰. Multiple auricular extrasystoles nodal rhythm sinus bradycardia and heart block are seen on occasion but are not of very serious importance. Multifocal ventricular extrasystoles however which may progress to ventricular fibrillation must be regarded seriously. Intravenous procaine has been used during the operation to reduce the incidence of arrhythmia^{11, 12} and other workers prefer intravenous injection of 100 mg doses of procaine amide¹. Procaine in any form is unfortunately a myocardial depressant and is not used as frequently now as it was a few years ago⁸. Should severe irregularity develop during the splitting of the valve the surgeon is asked to rest awhile until the insulted myocardium has regained its composure. Auricular fibrillation before the operation does not appear to increase the risk¹⁰. If the heart beat becomes weak during the operation the blood pressure can be raised by an intravenous drip of noradrenaline¹⁰ or phenyl ephrine¹³. Since coronary flow depends on the pressure of blood in the proximal aorta an adequate blood pressure must be maintained and deep anaesthesia avoided to ensure this. Additional measures taken to control arrhythmia include the pre operative oral administration of quinidine sulphate gr 9 and the injection of 5 ml of 4 per cent lignocaine into the pericardial sac¹⁰.

At the conclusion of the cardiac part of the operation the lung is re expanded and when the chest wall is closed the period of apnoea

the increase in heart rate of about 10 beats a minute, so caused. When relaxation of the cords is optimal a cuffed endotracheal tube is passed orally and is connected to a machine delivering a nitrous oxide oxygen mixture which may vary from 50 per cent of each to 75 per cent and 25 per cent. A closed circuit with a controlled leak is usually employed and controlled respiration is commenced. Some observers prefer assisted breathing.^{8 11} If the initial dose of relaxant does not cause apnoea, additional amounts must be injected. While this rapid induction with thiopentone and a relaxant has been proved to be relatively safe in many hundreds of cases there are experienced anaesthetists who prefer to use gas oxygen and ether⁸ for induction and intubation, afterwards running on minimal amounts of ether with assisted breathing or on gas oxygen and a relaxant. This method of anaesthesia is sometimes preferred when the patient is a young child or when the operation is for relief of constrictive pericarditis, as there is evidence⁹ that severe circulatory collapse may follow the injection of thiopentone in this last condition. Even when gas oxygen ether is used at the beginning the intubation can be made easy by injection of a relaxant rather than by increasing the ether concentration.

At least one reliable intravenous drip apparatus must be set up so that large volumes of blood can be quickly transfused in those rare conditions when it is needed.

The maintenance of anaesthesia demands above all else the provision of an adequate tidal exchange and this is effected either by manual bag pressure or by a mechanical respirator. The greatest depth of anaesthesia of the whole operation is needed for intubation so that straining and hypoxia are avoided. All general anaesthetics cause vasodilation and a decrease in peripheral resistance proportional to depth. This is normally compensated for by increased cardiac output but in mitral stenosis the cardiac output is relatively fixed. The minimal amount of anaesthetic which will abolish the cough reflex is all that is required. While some workers supplement the drugs used for induction by intermittent doses of pethidine (20 mg.), or thiopentone⁹ others use small quantities of ether.^{8 11} To reduce reflex activity during anaesthesia and to relieve post operative pain some workers favour a paravertebral block involving two nerves above and below the interspace of the incision. This can be done either by the surgeon or by the anaesthetist. It is occasionally done at the conclusion of the operation to lessen post operative pain¹ while other workers inject local analgesic solution into the periosteum of the excised rib.¹¹

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The control of arrhythmias¹⁶ is an important part of the management of cardiac operations and constant ECG monitoring is helpful. Irregularities of the heart may be due to the state of anaesthesia to vagal stimuli from the endotracheal tube to rib spreading and to manipulation of the heart¹⁰. Multiple auricular extrasystoles nodal rhythm sinus bradycardia and heart block are seen on occasion but are not of very serious importance. Multiple focal ventricular extrasystoles however which may progress to ventricular fibrillation must be regarded seriously. Intravenous procaine has been used during the operation to reduce the incidence of arrhythmia^{11, 18} and other workers prefer intravenous injection of 100 mg doses of procaine amide¹. Procaine in any form is unfortunately a myocardial depressant and is not used as frequently now as it was a few years ago⁹. Should severe irregularity develop during the splitting of the valve the surgeon is asked to rest awhile until the insulted myocardium has regained its composure. Auricular fibrillation before the operation does not appear to increase the risk¹⁰. If the heart beat becomes weak during the operation the blood pressure can be raised by an intravenous drip of noradrenaline¹⁰ or phenyl ephrine¹¹. Since coronary flow depends on the pressure of blood in the proximal aorta an adequate blood pressure must be maintained and deep anaesthesia avoided to ensure this. Additional measures taken to control arrhythmia include the pre operative oral administration of quinidine sulphate gr 9 and the injection of 5 ml of 4 per cent lignocaine into the pericardial sac¹⁰.

At the conclusion of the cardiac part of the operation the lung is re expanded and when the chest wall is closed the period of apnoea

is allowed to terminate. It is frequently necessary to reverse the residual action of the relaxant by the intravenous injection of atropine gr $\frac{1}{16}$ to $\frac{1}{8}$ followed by neostigmine 1-3 mg.

Hypotensive anaesthesia has a limited use in the surgery of the heart and great vessels and is of most help in operations for the relief of aortic coarctation. In these patients the chest wall bleeds very freely unless the blood pressure is low and hypotension also facilitates the actual suturing or grafting of the aorta. The pressure should be on the way up when the aortic clamps are removed so that the vascular bed in the abdomen and legs is not too large when the blood flows into it. If this precaution is not taken profound circulatory collapse may result.⁹ Some workers prefer to use hexamethonium to get their hypotension⁹ while others use trimetaphan camphosulphonate (arfonad).¹⁵

The very topical and intriguing question of induced hypothermia is considered in Chapter 12.

References

- 1 CAMPBELL, M., 1955 *Brit med Bull.*, 11 178 (Sept.)
- 2 WOOD PAUL, 1955 *Brit med Bull.*, 11 203 (Sept.)
- 3 O'DONNELL, J. A., and McDERMOTT T. F., 1955 *Anesthesiology* 16 343 (May)
- 4 REHN, L., 1897 *Arch klin. Chir.*, 55 315
- 5 SOUTTAR, H. S., 1925 *Brit med J.*, 2, 603
- 6 PRIERAM, B. O., 1926 *Arch klin. Chir.*, 142, 458
- 7 BAILEY, C. P., 1949 *Dis Chest* 15 377
- 8 PENDER, J. W., 1953 *Anesthesiology* 14 77 (Jan.)
- 9 BROWN, A. I. PARRY and SELICK, B. A., 1955 *Brit med Bull.*, 11 174 (Sept.)
- 10 BROWN, W. M., and REID, J. E., 1954 *Anæsthesia*, 9 68 (April)
- 11 SADOVE, M. ■ WYANT, G. M., JULIAN, O. C., and DYE, W. S., 1955 *Anesthesiology* 16 133 (Jan.)
- 12 PRESTON, F. S., 1953 *Brit J Anæsth.*, 25 299 (Oct.)
- 13 MASON, G. A., 1955 *Brit med Bull.*, 11 183 (Sept.) quoting Joan Miller
- 14 WARMUTH, C. E., 1954 *Anæsth. & Analges.*, 33 115 (March-April)
- 15 TUBBS, O. S., 1955 *Brit med Bull.*, 11 199 (Sept.)
- 16 SELICK, ■ A., 1956 *Proc World Congress of Anæsthesiologists* (1955) p 58 Minneapolis Burgess.

CHAPTER 17

ANÆSTHESIA AND ANALGESIA FOR ABDOMINAL SURGERY

It is not proposed to discuss anæsthetic techniques for routine operations upon the abdomen. In Great Britain the most usual method is maintenance of a light level of narcosis reinforced by a muscle relaxant. The use of small doses of chlorpromazine (q.v.) before and after operation seems to be beneficial in several respects e.g. reduction of nausea and vomiting with little if any added risk. A considerable amount of work has been carried out recently on several aspects of abdominal surgery and these will now be considered briefly.

Acute Intestinal Obstruction

Operations for the relief of acute intestinal obstruction are always an anxiety to the anæsthetist.² After premedication but before the induction of anæsthesia the nose and throat should be cocaineized and the largest possible œsophageal tube passed down into the stomach through the nose. A determined effort should now be made to empty the stomach by suction through this tube the position being altered and partial rotation carried out to avoid blockage of the distal end. This tube should be kept in position throughout and after operation suction being used intermittently as required. If no suction pump is available, an aural or bladder syringe or even a 20 ml. Record syringe can be pressed into service. Even after this procedure has been carried out it is wise to assume that the stomach is still not empty and the question arises as to the safest technique to avoid the inhalation of regurgitated fluid.

Spinal analgesia was at one time popular as it provides excellent relaxation and does not abolish the cough reflex. It must however, reach the level of T₅ and the muscle paralysis involved does impair the efficiency of coughing. Furthermore considerable circulatory depression will occur and this may prove fatal in a patient with an already low blood pressure due to imperfect filling of the heart from pressure on the great veins from abdominal distension.

The same remarks apply to a rather lesser extent to extradural block and this technique may be extremely difficult in a patient whose abdomen is greatly distended and who is in considerable pain and distress.

Local analgesia comprising mainly infiltration of the abdominal wall causes no appreciable fall of blood pressure nor does it impair coughing to any considerable extent but it is usually impracticable, as on opening the peritoneum the distended intestines are forced out and may be almost impossible to return later. A combination of infiltration with posterior splanchnic or posterior intercostal blocks may be feasible but a significant fall in blood pressure must be expected.

We are therefore led to the conclusion that for gravely ill patients some type of general anæsthesia should be employed. Until recently it was usually held that the induction of narcosis should be effected by an inhalation technique such as nitrous oxide-oxygen-ether, the theory being that if regurgitation should occur it will do so when the laryngeal reflex is still present so that rapid tilting of the patient into the head down position and suction will avoid the aspiration of intestinal contents. When adequate relaxation had been attained a cuffed endotracheal tube should be passed.

Recent work, however, tends to show that it is safer to abolish active vomiting directly unconsciousness supervenes and this can be done by giving an intravenous relaxant with, or immediately before an intravenous injection of a rapidly acting barbiturate (Hexobarbitone is possibly preferable to thiopentone in these circumstances as it causes less fall in blood pressure). The original idea that this procedure was liable to cause regurgitation owing to the relaxation of the cardiac sphincter is probably not entirely correct²¹ as this appears to act more as a valve than a sphincter and to be practically unaffected by relaxants. On the other hand if there is any obstruction to inspiration the cardiac valve becomes incompetent. It is therefore suggested that a rapid induction of anæsthesia should be carried out with an intravenous barbiturate and a relaxant with the patient in the greatest possible foot down tilt. In this way active vomiting is suppressed and the intra gastric pressure will not be high enough to force fluid against gravity up to the level of the pharynx unless extreme abdominal distension has occurred.² A cuffed tube is then passed and inflated and the patient levelled out to avoid a further fall in blood pressure. Anæsthesia is then maintained with nitrous oxide-oxygen and minimal ether or with cyclopropane. The inexperienced anæsthetist is warned that this technique of rapid intubation with a cuffed tube may present considerable difficulties in practice. A modification of the method has proved useful when the patient is so ill that an intravenous barbiturate might cause fatal hypotension. Anæsthesia is induced in the head up position by the

inhalation of a 50 per cent cyclopropane-oxygen mixture. Unconsciousness supervenes in a few breaths, and after the intravenous injection of an appropriate relaxant a cuffed endotracheal tube is inserted.

The question of fluid balance is always important when dealing with cases of acute intestinal obstruction but cannot be fully discussed here. If the block is high vomiting occurs early and will lead to dehydration, loss of chlorides and alkalosis. This will call for an intravenous drip infusion of saline. Low obstruction on the other hand is characterized by abdominal distension, deficient circulation in the bowel wall and a low blood pressure.

Upper Abdominal Surgery

When operating upon the upper abdomen many surgeons prefer to have the patient's arms abducted on a double splint. This is a convenient position and does not impede respiration, but it is necessary to observe three precautions. The splint should not be quite straight so that the arms are abducted to slightly less than a right angle. The splint should be padded with sorbo rubber two inches thick except where it passes behind the mattress on the operating table so that the patient's arms are not forced backwards. Finally the hands should be pronated rather than supinated and the wrists secured by a loose bandage or by a rubber tube and artery forceps. In right handed patients an intravenous drip can, if necessary, be set up in the left forearm and the blood pressure recording cuff fitted to the right arm. One of the writers has anaesthetized over 2000 patients in this position without neurological complications.

Before operations on the stomach it is usual to pass a small gastric tube through the nose and the anaesthetist can apply intermittent suction to this so that fluid or gas in the stomach will not impede the surgeon's work.

Traction on the viscera immediately below the diaphragm may cause a fall in blood pressure and laryngeal spasm (the Brewer Luckhardt reflex) and for this reason alone endotracheal anaesthesia is advantageous. An inflated cuff on the tube will also prevent any possibility of the inhalation of gastric fluid and will facilitate assisted or controlled respiration.

During operations on the biliary tract it is now becoming common to take cholangiograms. In this event natural respiration should be entirely abolished and full control instituted so that during the exposure the abdominal viscera are motionless and a clear picture should result.

Removal of Phæochromocytomata and Bilateral Adrenalectomy

In the past few years considerable progress has been made in the diagnosis and removal of these tumours. Several facts should be borne in mind when considering the anæsthetic technique to be used. In the first place when they are handled phæochromocytomata release large quantities of sympathomimetic amines—mainly noradrenaline but to a lesser extent adrenaline—so that there may be considerable hypertension during the initial stages of the operation.¹ This can be controlled by the intravenous injection of successive doses of from 3 to 5 mg of phentolamine (regitine rogatine C 7337) which is an idrenolytic agent² with the following chemical structure, 2 (N p tolyl N m hydroxyphenyl aminomethyl)-imidozolinemethanesulphonate. Secondly the excess of sympathomimetic amines in the circulation renders it inadvisable to employ anæsthetic agents capable of producing ventricular arrhythmias in such circumstances, such as cyclopropane and chloroform. Finally when the venous return from these tumours has been cut off, hypotension may develop and this appears to be combated most efficiently by hydrocortisone or a noradrenaline drip infusion (4 mg per litre in 5 per cent glucose) so regulated that the systolic pressure is kept above 100 mg Hg.⁴ The drip may have to be continued after the patient has been returned to bed and then gradually reduced.

Bilateral adrenalectomy has recently become a fairly common operation for retarding the growth of metastatic tumours originating in the breast and prostate. The blood pressure changes described above are rarely noticeable on the operating table although marked hypotension may be a feature of the post operative period. The chief hazard from the anæsthetic point of view is occasional massive hæmorrhage from an abnormal vessel which may prove difficult to control. For this reason it is always wise to set up an intravenous infusion at the beginning so that blood can be given without delay.

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The Trendelenburg Position

The head down position in surgery was originally described by W Meyer in 1885⁵ and it was not until 5 years later that Trendelenburg himself recommended a 45° slope in order to render the interior of the pelvis accessible to the surgeon.⁶ Since then the Trendelenburg position has been in constant use in gynæcological operations and it behoves the anæsthetist to consider its implications.

In a young healthy subject a head down tilt has little effect on the blood pressure and cardiac output.⁷ Unfortunately many patients are elderly and have some form of vascular disease so that their

vasomotor control is impaired. Furthermore many drugs used in the anæsthetic technique cause partial paralysis of the sympathetic system. For example this may occur in deep general narcosis, with ganglion blocking agents such as the derivatives of methonium and thiophanium, with chlorpromazine and with peridural and sub-arachnoid blocks. Under these conditions vasomotor control will be inefficient and since in a head down tilt most of the capillary blood is above instead of below heart level, increased cardiac filling will occur with consequent rise in blood pressure, venous pressure and stroke volume.⁸ For a short time these effects will be beneficial to shocked patients but the induced hypertension may have serious consequences such as retinal detachment⁹ in patients whose normal blood pressure is high. In passing it should be noted that in a head down tilt, the blood pressure will vary with the part of the body being considered, being 2 mm. Hg higher or lower than that at heart level for every vertical inch below or above that level.¹⁰

The Trendelenburg position affects the respiratory system mainly by raising the diaphragm which not only has the movable contents of the abdomen resting upon it but often an abdominal pack and retractor as well. As a result the vital capacity is considerably lowered,¹¹ the decrease being proportional to the angle of tilt.¹² If natural breathing is allowed to persist increased respiratory efforts will occur which tend to nullify the objects of the position from the surgeon's standpoint. Some degree of hypoxia and hypercarbia will also develop which besides being unphysiological will tend to cause congestion and oozing from cut surfaces. If muscle relaxants are used the diaphragm will lose its tone and will be pushed passively still higher up the thorax. On the other hand relaxants facilitate the use of assisted or completely controlled respiration and are usually indicated in long gynæcological operations. Every effort must be made to avoid a build up of carbon dioxide, but some increase may be inevitable especially in high degrees of tilt.¹³

Two other details in the anæsthetic technique may be mentioned. It is often worth while passing a small stomach tube and sucking out any liquid or gaseous gastric contents. A completely empty stomach gives more room for the intestines packed into the concavity of the diaphragm. Secondly gastric juice, saliva and mucus tend to collect in the nasopharynx and should be removed by suction before the operating table is straightened out at the end of the operation.

The method of maintaining the Trendelenburg position is a matter of considerable concern to the anæsthetist. One early technique was to bend the lower part of the operating table at the

level of the patient's knees and to strap the legs to the bent flaps. Considerable strain on the knee joints ensued and the venous return through the calves was impeded. The incidence of post operative venous thrombosis and pulmonary embolism was almost certainly increased.

Padded shoulder rests were next introduced and are still in common use. However carefully they are adjusted, the brachial plexus can either be compressed or stretched with the risk of subsequent palsy. This complication is usually transient but may be permanent with the probability of future legal actions. Curved shoulder rests padded with thick sorbo rubber were designed to spread the pressure over a wider area but they were not entirely satisfactory.

Pelvic rests were next tried to take the patient's weight on the brim of the pelvis. Two patterns were evolved in this country and attained some vogue, but they tend to impede the surgeon by bunching up the anterior abdominal wall and are difficult to adjust so that slipping is impossible.¹⁴

The problem remained unsolved¹⁵ until advantage was taken of skin friction spread over a very wide area of the patient's back and legs.¹⁶ A sorbo rubber mattress ribbed on its upper surface is laid on the operating table and secured by wide metal hooks to the foot end.¹⁷ Three movable ribbed bolsters are fitted into the concavities of the neck, lumbar spine and Achilles tendons¹⁸ as shown in the illustrations. When the table is tilted the corrugations of the bolster fit into those of the mattress giving an interlocking and cog wheel effect which holds the patient firmly in position. The centre bolster is narrower than the other two and the patient's arms are secured to it by straps. As the normal lumbar curve is retained post operative backache is considerably diminished. The small bolster under the Achilles tendons keeps weight off the calves and this should reduce the likelihood of venous thrombosis. It has been found in practice that if this method is properly applied with no clothing between the patient's skin and the rubber corrugations no slip occurs even at the maximum tilt (50°) possible with most operation tables.

Whatever method of maintaining the patient in position is used the minimum tilt consistent with the surgeon's needs should not be exceeded and it should be restricted to the shortest possible time. When the table is levelled out this should always be done gradually and a careful watch should be kept on the blood pressure and pulse rate.

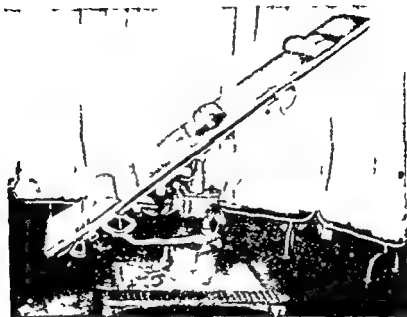


FIG 64 Ribbed mattress with 3 bolsters (Hewer *Anæsthesia*)

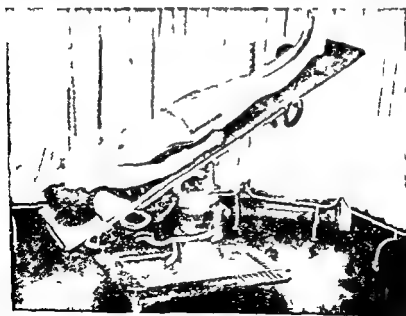


FIG 65 Patient in 45° head-down tilt on ribbed mattress (Hewer *Anæsthesia*)

Synchronous-combined Abdomino-perineal Operations

Simultaneous operations by two surgeons from abdominal and perineal incisions are often performed for removal of the rectum or pelvic exenteration for malignant growths

The position of the patient is an awkward one, being a combination of the lithotomy and Trendelenburg postures. The usual shoulder rests on extensions can be dispensed with if the ribbed mattress already described is employed. A short divided mattress is available but a standard length one can be used if the superfluous length at the head end of table is allowed to hang down towards the floor (Fig. 66)

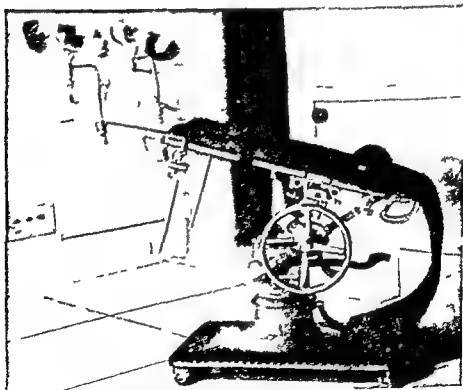


FIG. 66. Operating table in position for synchronous-combined operation. If a divided ribbed mattress is used the portion hanging down is eliminated. (Dept of Medical Photography St Bartholomew's Hospital.)

A point to observe is that the anaesthetist tends to be preoccupied with the abdominal wound which he can see and does not realize that much more bleeding is probably taking place from the perineal

wound which is invisible to him. It is essential to set up a satisfactory blood transfusion at the beginning of the operation and to replace blood as it is lost. The writers have found that the forearm is the most satisfactory site for the infusions. The patient's wrists are strapped to his thighs and extra long tubing enables the drip chamber to be placed near the head of the table under the anaesthetist's control.

Urethral Instrumentation

Although not, strictly speaking, an abdominal operation, local analgesia for the passage of bougies and catheters and cystoscopy can be conveniently considered here. Up to the present time very indifferent results have occurred and much pain and distress have been experienced by patients.

A preparation of 2 per cent lignocaine jelly known as "xylocaine gel" is most useful for urethral analgesia.⁹ In males, the bakelite nozzle supplied with each tube is inserted into the meatus and the contents expressed gently into the urethra. As the nozzle is removed the penis is compressed with an elastic band to prevent leakage and the jelly is then massaged into the posterior urethra. In from 10 to 15 minutes instrumentation can be started. In females a throat swab dipped in xylocaine gel is inserted into the urethra and kept there for 10 minutes. The results appear to be much better than those obtained with traditional techniques.

Relief of Pain after Abdominal Surgery

It is often difficult to estimate the amount of pain being experienced by a patient after an abdominal operation and the effect of any analgesic drugs which may have been given. It has recently been pointed out that under these conditions, pain is more or less proportional to the decreased vital capacity compared with that before operation. If therefore spirometry is carried out the effects of analgesic drugs or other pain relieving measures can be calculated with considerable accuracy.¹⁰

References

- 1 McLEAN J. A. 1953 *Brit med J* Jan 24 201
- 2 PAPPER E. M. and CAMILL, G. F. 1955 *J Amer med Ass* 157 309
- 3 HELPS E. P. W. *et al* 1955 *Lancet* Aug 6 267
- 4 KNOX J. and SLESSOR A. 1955 *Lancet* April 16 790
- 5 MEYER, W. *Arch klin Chir* 31
- 6 TRENDLENBURG F. 1940 quoted in *Medical Classics* 4 936
- 7 GROLLMAN 1932 *Cardiac Output of Man in Health and Disease* London
- 8 GORDH T. 1945 *Postural Changes* Stockholm
- 9 DALY A. 1944 personal communication

- 10 ENDERBY G E. H., 1954 *Lancet* Jan. 23 185
- 11 ALTSCHULE, M. D., and ZAMCHECK, H. 1942, *Surg Gynec Obstet.*, 74 1061
- 12 CASE, E. H., and STILES J A., 1946 *Anesthesiology* 7 29
- 13 LUCAS, H. C. B., and MILNE, E. H., 1950 *Proc R. Soc Med (An. Sec)* Jan. 2.
- 14 { WARD R. O., 1950 *Lancet* 1 423
HANS S F., 1952, *Lancet* 2, 669
- 15 { HOWKINS J., 1952, *Lancet* 2 759
HEWER, C. L., *Lancet* 2, 826
- 16 HEWER, C. L., 1953 *Lancet* March 14 522
- 17 { HEWER, C. L., 1954 *Med Annu.*, 39
HEWER, C. L., 1956 *Canad Med Ass J* Feb 15 285
- 18 { HEWER, C. L. 1953 *Anæsthesia* July 198
HEWER, C. L. 1955 *Brit med. J.*, July 9 127
- 19 BROMAGE, P R., 1955 *Brit med J* Sept 3 589
- 20 BRYCE SMITH R., 1955 *Brit med J.*, Feb 19 462.
- 21 O MULLANE, E. J., 1954 *Lancet* June 12, 1209
- 22 MORTON H J V., and WYLIE, W. D., 1951 *Anæsthesia* Oct., 190

ANÆSTHESIA IN OBSTETRICS

Apparatus for Self-administration of Trilene—Dangers of Vomiting — Pudendal Block — Subarachnoid Block — Extradural Analgesia — Caudal Block — Muscle Relaxants — Analgesic Drugs — Eclampsia — Placental Transmission — Phenothiazine Drugs — Obstetric Shock — Hypofibrinogenæmia — Breech Delivery— Infant Resuscitation — Retrolental Fibroplasia

The Use of Trichlorethylene by Midwives

THE MINISTER of Health has now authorized the use by midwives of trichlorethylene and the Central Midwives Board has approved the following machines for its administration. The Tecota Mark 6 made by Messrs Cyprane Ltd and the Emotril Trichlorethylene Automatic Inhaler made by Medical and Industrial Equipment Ltd. The Board stipulates that the apparatus shall be used by a midwife on her own responsibility only if it has been inspected and approved by the Board within the previous six months. The apparatus must be inspected periodically by the manufacturers who will arrange with the Board for the issue of the requisite certificates. Before employing the apparatus each midwife must satisfy the Board that she is thoroughly proficient in its use. The new rules provide that midwives shall from time to time attend refresher courses approved by the Board. Midwives are already allowed to use pethidine on their own responsibility.

The Dangers of Vomiting

The administration of general anæsthetics to patients in labour is associated with a definite mortality, the mechanism of which is usually the inhalation of stomach contents into the bronchial tree. Labouring women seldom have an empty stomach so that great care is necessary during the stage of induction. They should only receive fluids and semi solids during labour. A simple, though not a foolproof method is to use nitrous oxide oxygen, trilene and ether with the addition of a little carbon dioxide to provide the urge to breathe and to help to carry the patient down into the third stage as rapidly as possible. Induction of anæsthesia with the patient on her side and the use of a transparent mask which is not strapped on by a harness are additional helps. Another technique is to raise the head and shoulders of the patient in order to prevent passive

- 10 ENDERBY, G E II 1954 *Lancet* Jan 23 185
- 11 ALTSCHULE M D and ZAMCHECK H 1942, *Surg Gynec Obstet.*, 74
1061
- 12 CASE, E II and STILES J A 1946 *Anesthesiology* 7 29
- 13 LUCAS II C II and MILNE, E II 1950 *Proc R Soc Med (An Sec)*
Jan 2
- 14 { WARD R O 1950 *Lancet* 1 423
HANS S F 1952 *Lancet* 2 669
- 15 { HOWKINS J 1952 *Lancet* 2 759
HEWER C L *Lancet* 2 826
- 16 HEWER C L 1953 *Lancet* March 14 522
- 17 { HEWER C L 1954 *Med Annu* 39
HEWER C L 1956 *Canad Med Ass J* Feb 15 285
- 18 { HEWER C L 1953 *Anæsthesia* July 198
HEWER C L 1955 *Brit med J* July 9 127
- 19 BROMAGE, P R 1955 *Brit med J* Sept 3 589
- 20 BRYCE SMITH R 1955 *Brit med J* Feb 19 462
- 21 O MULLANE E J 1954 *Lancet* June 12 1209
- 22 MORTON II J V and WYLIE W D 1951 *Anæsthesia* Oct 190

A labour ward bed for safer anaesthesia has been described by Gibberd¹. It enables the head of the patient to be lowered immediately vomiting takes place so that postural treatment can be adopted before the vomit can be inhaled. In this bed, the upper surface is divided into three equal sections. One side of the middle section pivots between the adjacent top and bottom sections while the other side of the middle section is free to fall below the level of the other two sections, thus giving the patient's whole trunk a head down inclination when she is in the lithotomy position across the bed. If it becomes necessary to tilt the patient, a sharp kick on the pivoted leg sends it under the bed and the middle section immediately drops into a tilted position.

A modification of the Oxford labour ward bed has been in use in at least one large hospital for over a year and has given great

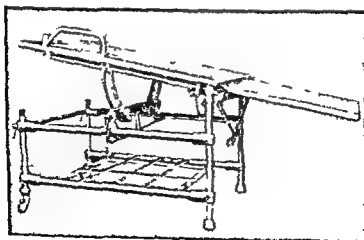
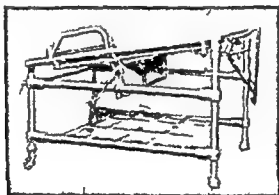


FIG 28 Modified Oxford labour ward bed (Wylie *Lancet*)

regurgitation of stomach contents and give a 50 per cent mixture of cyclopropane in oxygen from the start. After twelve to twenty breaths, when anæsthesia is present an intravenous injection of suxamethonium is given and a cuffed endotracheal tube inserted and the cuff inflated.²¹ Maintenance then presents no problem. The insertion of a 12 gauge œsophageal tube or a number 20 Wangensteen stomach tube before induction of anæsthesia is a safety measure but opinions vary as to the advisability of using it in every case where a full stomach is suspected. A stormy induction is, after a full stomach, the most common cause of vomiting, and it may well be that induction with an intravenous thiobarbiturate if the dose is not in excess of 250 mg, is the readiest means of securing a smooth and undisturbed anæsthesia.²² Cyclopropane can then be used for maintenance.

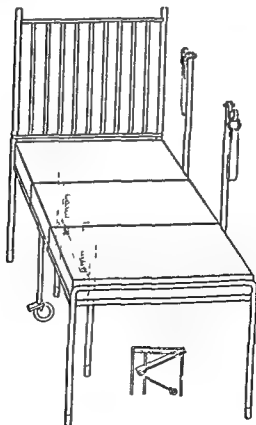


FIG 67 A labour ward bed (Gibberd *Lancet*)

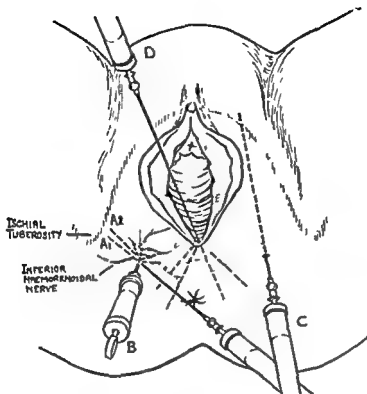


FIG 69 Pudendal block (Gate and Dutton *Brit med J*)

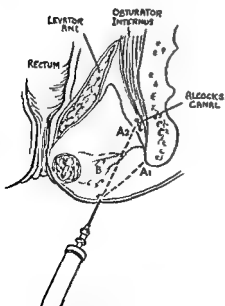


FIG 70 Pudendal block (Gate and Dutton *Brit med J*)

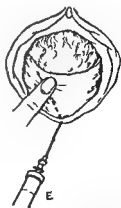


FIG 71 Local infiltration of perineum (Gate and Dutton *Brit med J*)

satisfaction to all concerned.²³ The whole bed can be tipped 20 degrees either head down or foot down in three seconds. The latter position will help in the prevention of regurgitation before intubation with a cuffed endotracheal tube during the application of the cyclopropane, muscle relaxant technique.⁴

Parker has again recently drawn attention to the dangers of inhalation of vomit in obstetric cases.²⁴ A syndrome described by Mendelsohn²⁵ may result from the aspiration of acid gastric contents and may not show itself for several hours after the occurrence of the accident. Then the onset of the illness is dramatic and the patient becomes extremely ill with cyanosis, dyspnoea and tachycardia, and shows the signs of acute pulmonary oedema with patchy consolidation seen on the skiagram and crepitations heard at the lung bases. Bronchospasm and asthma may or may not be seen.⁴ This syndrome appears to be confined to obstetric patients who inhale acid gastric contents although surgical patients who are subjected to the same insult do not seem to develop it. Reasons have been given⁴ for assuming that the underlying cause is acute suprarenal insufficiency associated with the state of parturition and successful results have followed the intravenous injection of 100 mg. of hydrocortisone, together with antibiotics and oxygen therapy. That aspiration of vomitus is a real danger is shown in an analysis of a series of 913 707 live births.²⁶ In this large number, forty five deaths were stated to be directly due to anæsthesia of which 29 per cent were ascribed to inhalation of vomit and 25 per cent to spinal analgesia. Twenty two per cent of these anæsthetic deaths could not be accounted for. This fear of inhalation of vomitus has led anæsthetists to adopt other means of pain relief so that local analgesia in one of its many forms is becoming the routine followed in an increasing number of clinics.

Pudendal Block

Pudendal block with or without local infiltration of the vulva, is a satisfactory form of analgesia for the outlet forceps operation and also for many cases of normal delivery.^{6, 7} The indications are said to include²⁷ fetal distress, delayed second stage, assisted breech delivery, multiple pregnancy and repair of severe lacerations. The technique does not provide good relaxation if intra uterine manipulations become necessary. Gate and Dutton⁶ have used local analgesia in 62 per cent of a consecutive series of forceps deliveries, some of them being mid forceps cases. Nervousness alone was not regarded by them as a definite contra indication but the

nupercaine in 2 ml of 10 per cent dextrose. The block is not given to patients with a systolic blood pressure of less than 100 mm of mercury. The blood pressure should be taken at five minute intervals. In this series 81 per cent were totally effective and headache was complained of in 21 per cent of the patients, but in only 2 per cent was it severe. In Britain a large series was reported by Thorne¹² with excellent results and no neurological complications and this has now been enlarged to cover over 800 cases.¹³ He uses hyperbaric nupercaine solution 0.8 ml for forceps delivery and 1.4 ml for Cæsarean section. Methylamphetamine is required by half the patients in the latter group. While the suspicion of spinal analgesia focused by the Woolley and Roe case¹⁴ still colours the view of many people on this useful method it must also be recalled that so experienced an observer as Macintosh¹⁵ has said that he does not believe a patient has any more tendency to develop paralysis after spinal analgesia than she has to die say after thiopentone, provided that all due care is taken.

Many methods of treating post spinal headache have been described. Its incidence has been reduced from 9 per cent in a control series to 3 per cent in a series treated by the injection of 15–20 ml of normal saline into the extradural space immediately following the subarachnoid block.¹⁶ (See also Chapter 9)

Lumbar Extradural Block

Extradural analgesia has its advocates for Cæsarean section. In a recent series of 200 successful cases¹⁷ the continuous technique was employed, the catheter being inserted with the patient in the sitting position. She is then lowered and a test dose of 2 ml of 2 per cent lignocaine is injected and is followed by the main dose of 10–15 ml of the same solution. Occasionally an additional 8 ml is needed after fifteen minutes. The use of 1.5 per cent solution would increase the safety of this technique. The blood pressure is controlled by an intravenous drip of noradrenaline. Another series reports 246 successful cases¹⁸ using 2 per cent cyclaine (hexylcaine) which is said to be less toxic than lignocaine of the same strength. The continuous technique is used, 15 ml of solution being the initial injection. In this series of patients hypertension was not regarded as a contra indication nor was hypotension unless due to shock or hæmorrhage. The technique was not employed in severely anæmic women. Another similar series gave results of equal satisfaction.¹⁹

method was avoided when vaginal examination suggested that the rotation of the occiput or the actual extraction of the head was likely to be attended with considerable difficulty. The method they describe is a combination of pudendal block and infiltration and gives satisfaction to the patient in 86 per cent of cases. If the method is limited to low forceps deliveries the success rate approaches 100 per cent. From a point midway between the ischial tuberosity and the anus and with one finger in the vagina or rectum, solution either 0.5 per cent procaine or 0.5 per cent lignocaine without adrenaline, is deposited bilaterally in the following sites: in the vicinity of the pudendal nerve as it traverses Alcock's canal on the lateral wall of the ischio-rectal fossa, among the branches of the inferior hemorrhoidal nerve which pass medially across the ischio-rectal fossa to the external anal sphincter and the skin between the anus and the ischial tuberosity. Solution is also deposited beneath the skin covering the lateral aspect of the labium majus as far anteriorly as the pubis, beneath the skin of the perineal body including the line of the episiotomy incision and beneath the mucosa of the posterior and lateral walls of the vagina in its lower third. A lumbar puncture needle 12 cm long is used for the deeper injections and about 80 ml of solution are used. Hyalase may be added to the solution but adrenaline sometimes inhibits labour pains and is better avoided. An interval of five to ten minutes should elapse between the finish of the injection and the application of the obstetric forceps. The details of technique are well set out in Gate and Dutton's paper⁸ and also by Klink.⁹

Subarachnoid Block

Spinal analgesia in obstetrics is still the subject of controversy. It has been recommended¹⁰ as the method of choice in patients having a full stomach, in patients who are heavily sedated, for the delivery of premature babies because of the absence of foetal respiratory depression and the good perineal relaxation produced, for patients with heart disease, for Cæsarean sections and for all patients who wish to be awake during and share in the experience of parturition. Block of the sacral nerves is referred to in the United States as saddle block and is there widely used. In a series of 800 cases¹¹ recently reported the results were good. With the patient sitting 2 ml of 0.2 per cent amethocaine in 10 per cent dextrose solution were injected through a small 22 or 24 gauge needle. The patient was then placed supine with a double pillow under the shoulders and the legs in stirrups. Other workers have used 30-50 mg of procaine or 3 mg of

in any of these cases. In another group, gallamine was the relaxant chosen²¹ for four hundred forceps deliveries and two hundred Cæsarean sections. For the former cases induction was by cyclopropane or gas oxygen and trilene and just before delivery 100 mg of gallamine was injected and anaesthesia maintained with gas and oxygen. For Cæsarean section induction was by thiopentone 250 mg or kemithal 500 mg, and maintenance by gas oxygen or cyclopropane. Gallamine 100 mg maintained good relaxation caused no depression of the baby or excessive bleeding gave good operating conditions and caused no increase in uterine tone. Neostigmine 2.5 mg and atropine were given if necessary at the end of the operation while atropine alone was used for premedication. The technique gives a quick pleasant induction with a safe and quiet maintenance but does not abolish the risk of aspiration of stomach contents. Thiopentone with suxamethonium or 100 mg of gallamine is usually satisfactory for external version followed by atropine and neostigmine if gallamine is employed.

Analgesic Drugs

The search for new analgesic drugs is never ending one of the more promising to have been developed in recent years being Nisental (alphaprodine). It should only be given when labour pains are well established and occurring at regular intervals. The initial dose is 40–80 mg with the majority of patients receiving 60 mg.⁶ This causes warmth contentment and sleep for two to three hours when the dose may have to be repeated. Nausea giddiness and vomiting are occasional complications.⁷ No interference with labour has been reported. It would appear that nisental relieves anxiety and causes calmness and a relaxed mental state but amnesia is not a feature. It is more rapidly acting especially if injected intravenously²⁸ of shorter duration and less potency than pethidine and is especially useful in labours which are not expected to last long. Its effects are enhanced when it is combined with scopolamine 0.4 to 0.6 mg.⁹ but some degree of foetal respiratory depression is associated with its use varying from 5 per cent²⁶ to 16 per cent.⁸ No foetal deaths have been reported as due to its employment nor does it cause serious undesirable side effects in the mother.

Pethidine either alone or in combination with scopolamine remains one of the most useful drugs which the obstetrician has at his disposal for the relief of pain in labour. The drug can be given by continuous intravenous drip²⁰ once active labour has started and so far no foetal or maternal ill effects have come to light. In

Continuous Caudal Block

Continuous caudal analgesia in the management of disordered uterine function in labour has been extensively used by Johnson^{20 21} The idea of the block is to paralyse both the motor and sensory fibres of the pelvic floor and the perineum together with the visceromotor parasympathetic connections to the cervix. The upward extension of analgesia to the upper border of the eleventh thoracic segment in the extradural space interrupts the sensory connections of the fundus uteri which enter the cord with the rami communicantes at this level, so that the rhythmic pains of labour are not felt. The sympathetic motor fibres to the body of the uterus are given off from the spinal cord above this level and so are not influenced by caudal analgesia although the opposing parasympathetic fibres from the sacral plexus are interrupted. Successful caudal block therefore secures unopposed and painless contraction of the body and fundus of the uterus acting upon a completely relaxed and insensitive cervix and lower birth canal and has been successfully used in dealing with the problem of either uterine inertia or cervical dystocia or a combination of these two conditions. The technique used in Johnson's cases followed the early description of Hingson and Edwards² in which 1.5 per cent. metycaine in Ringer's solution and a malleable needle were employed. 1.5 per cent. xylocaine gives rather better results. The initial dose of 40 ml. is supplemented at about half hourly intervals by 15 ml. injections. In this series continuous caudal analgesia accelerated the progress of labour in a high proportion of cases and considerably decreased the foetal hazards of what to the obstetrician can be a perplexing problem.

Muscle Relaxants

Muscle relaxant drugs have been employed in obstetrics as in all other branches of anæsthesia and with considerable benefit both to the patient and to her medical attendant. They are used to provide relaxation of the perineum and abdominal wall and to facilitate intubation. There is evidence that 11 tubocurarine enters the foetal circulation much less readily than does gallamine.²⁶ Most of the relaxants in common use have been successfully given to labouring patients and a series of 225 cases is described²³ in which 2 mg. of dca methonium was injected, with the patient lightly anæsthetized with gas oxygen and trichlorethylene or with cyclopropane when the foetal head distended the perineum. The ensuing forceps delivery was made easier by the relaxed perineum. Occasionally a second dose of the relaxant was needed. No effects on the baby were noted

follows delivery. The method is not recommended for routine use in all cases of eclampsia,³⁵ but for grave cases might well be tried more often. Heavy sedation is not required in patients so treated and both maternal and foetal mortality are favourably influenced.

The pitocin drip and the use of this drug in any form in labour—or in general surgery—carries a risk if at the same time anaesthetics are used which may cause irritability of the automatic conductive tissue of the heart. The death is reported³⁶ of a patient who received a pitocin drip before she had been allowed to rid herself of cyclopropane used for induction of anaesthesia.

The So-called Placental Barrier

The barrier provided by the placenta to the transference of drugs from the maternal to the foetal circulation has been the subject of experimental investigation.³⁷ The evidence for assuming the placental transmission of muscle relaxants has been in the past rather confusing. Pittinger and Morris³⁸ were able to demonstrate placental transmission of tubocurarine following its injection into the uterine artery of dogs but were unable to show clinical curarization in human babies delivered with the aid of therapeutic doses of the relaxant. Decamethonium was shown to cross the barrier following the intravenous injection of large doses just before delivery.³⁹ Suxamethonium has been reported to pass the barrier by one observer⁴⁰ but this was not the opinion of another.⁴¹ Placental transmission of gallamine and suxamethonium has been demonstrated in dogs³⁷ by injection into the uterine artery, whereas decamethonium did not pass the barrier when the same technique of injection was used. In a recent series of cases⁷⁶ it was shown that when equipotent doses of d tubocurarine and gallamine were used before delivery the former appeared in the foetal serum in traces only while gallamine was quite often found in readily detectable and possibly significant amounts. In spite of these conflicting reports it would seem a safe and reasonable thing to use therapeutic doses of any of these relaxants other than perhaps gallamine as part of the anaesthetic technique for obstetrical delivery.⁴ It has of course been shown that anaesthetic gases vapours barbiturates paraldehyde chloral hydrate morphine and pethidine are all transmitted from mother to foetus.⁴²

The placental transmission of thiopentone has been carefully investigated by McKechnie and Converse⁴⁴ who found that thiopentone administered to obstetrical patients immediately prior to delivery crossed the placental barrier rapidly appearing in the

the early stages of labour a barbiturate will give relief and when dilatation of the cervix is complete, pethidine 50 to 100 mg with scopolamine gr $\frac{1}{16}$ to $\frac{1}{8}$ can be given intravenously slowly, and is said³¹ to be sufficient for forceps delivery and perineal repair.

In a careful analysis of 7000 patients, Hingson and Hellman³ came to the conclusion that the most successful routine for pain relief in labour was 100 mg of pentobarbitone intravenously at the beginning of active labour together with 100 mg of seconal by mouth and 100 mg of pethidine intramuscularly with 0.2 mg of scopolamine given with the pethidine and repeated two hourly.

Chloral hydrate is still a popular hypnotic in both institutional and domestic midwifery. It has recently been shown³² that soon after the oral administration of 1.4 to 3.1 g its presence can be demonstrated in both the maternal and the foetal blood streams while its elimination takes eight hours. Some of the drug is oxidized to trichloroacetic acid and some of it reduced to trichlorethanol. No ill effects on either mother or child were demonstrated.

Eclampsia

The anaesthetist is sometimes asked by his obstetrical colleagues to advise and help in the treatment of eclampsia. In a series of 130 cases recently reported³³ the eclamptic patient was given 4 ml of bromethol in five ounces of distilled water by drip into the rectum and when sedated one pint of 20 per cent dextrose was infused intravenously. To this drip were added 10 ml of 10 per cent calcium gluconate and a concentration of B and C vitamins. The rectal drip of bromethol was repeated in five hours and even a third time if necessary and was found to be a reliable sedative and anti-convulsant in eclampsia. In this series the maternal mortality was 5.4 per cent and the foetal mortality 11.2 per cent. Continuous spinal analgesia gives good results³⁴ the initial injection consisting of 0.5 ml of 1.5 per cent metycaine solution which is repeated each fifteen minutes as required. A catheter must be used and in most cases the blood pressure drops satisfactorily after the first injection. Continuous caudal analgesia is disappointing in the treatment of eclampsia but continuous extradural block is a promising method of management.³⁵ As the conduction block may have to be maintained for several days at a time the methods taken to prevent infection while the plastic tubing is in the extradural space must be meticulous. The blood pressure can be fairly well controlled and if labour is retarded a pitocin drip should be set up to stimulate uterine contractions. Oliguria is not directly relieved but a diuresis

25 mg doses by mouth or intramuscularly, repeated if necessary once or twice daily chlorpromazine is of definite value to the great majority of patients complaining of nausea and vomiting during pregnancy.⁴ Drowsiness may be a side effect but can be controlled by dextroamphetamine sulphate 5-10 mg. It has no harmful actions on either mother or child, used in this way. It seems to give excellent psychic sedation when given intravenously in 25 mg doses to patients in labour causing them to sleep between pains but to be rousable⁴⁸ and free from apprehension. The best results are likely to follow administration of pethidine early in labour and chlorpromazine just before full dilatation in primigravidae and at half dilatation in multigravidae. For the actual delivery the need for general anaesthesia is reduced and gas and oxygen alone frequently suffice.⁴⁸ Vomiting during labour is relieved and no untoward effects on either mother or child have been noted. The use of the drug may be indicated in premature delivery because of the absence of narcotizing effect on the baby. It is said to produce a medical leucotomy so that the patient bears pain better and is less worried by it.⁴⁹ A slight fall in blood pressure usually follows its injection.⁴⁹ On the other hand Savage⁵⁰ showed in a series of 127 cases that the drug gave no significant pain relief when compared with a control group who did not receive the drug. With increasing doses uterine inertia was the price paid for better pain relief and an increase in the forceps delivery rate and a prolongation of labour resulted in primigravidae. He also found that nitrous oxide air mixture was not potentiated by chlorpromazine. The third stage of labour was not prolonged. Savage as a result of his investigations advises against the routine use of this drug in labour. (See also Chapter 11.)

While chlorpromazine is rather unpredictable in its actions, promethazine used during labour together with pethidine is reported⁵¹ to offer the mother more effective sedation and her baby less harm than any other combination of drugs. When the need for sedation becomes apparent 25 mg of promethazine and 50 mg of pethidine are injected intramuscularly and repeated if necessary. The patients become sleepy but remain mentally co-operative and alert while the duration of labour is shortened. No ill effects on either mother or child were seen in this group of 300 cases.⁵¹

The phenothiazine drugs have been successfully used in the management of eclampsia.⁵² Diuresis is produced and the blood pressure falls during treatment. Small doses of oxytocin prevent uterine inertia and delayed delivery.

mixed foetal cord blood in 45 seconds and reaching an equilibrium with maternal venous blood in 3 minutes. It was unusual for babies so delivered to suffer from respiratory depression. No correlation between the amount and depth of anæsthesia, foetal responsiveness and thiopentone concentration in mixed umbilical cord and maternal venous blood was evident in this series of experiments. The lack of correlation may be explained by postulating that foetal liver tissue is more adept than adult liver in detoxicating thiopentone. More likely is it that thiopentone is distributed from the maternal blood stream to the maternal tissues so rapidly that the actual quantity of drug reaching the infant is not enough to cause cerebral depression. Over a longer period of time it seems probable that if repeated injections of thiopentone are given, a greater percentage of each succeeding dose will reach the infant as the maternal tissues approach saturation level and narcosis of the infant will result. The authors thus recommend that for vaginal delivery, thiopentone should be restricted to a single minimal sleep dose given just before delivery, while for Cæsarean section it should be limited to the induction period.

A similar conclusion has been reached by Crawford² who showed that there is no appreciable placental barrier to thiopentone and that the foetal blood level of the drug is at a maximum at the onset of anæsthesia and thereafter falls. He found that the longer the time interval between the injection of thiopentone and the delivery of the child the lower the foetal blood thiopentone level is likely to be. He regards 250 mg used for induction of anæsthesia but not repeated to be reasonably safe.

The passage of ether from mother to foetus in experiments on animals⁴⁵ was investigated and it was shown that ether concentrations were almost the same in the brain and liver of the foetus as in the placenta, and considerably lower than in the arterial blood and brain of the mother. The ether concentration in the foetus rose at approximately the same rate as that in the maternal muscles. Oximetric studies of the blood of neonates during the first thirty minutes of life show⁴⁶ that pethidine with scopolamine given to the mother cause a slight depression in blood oxygen saturation but are not likely to be harmful. A similar experiment using trichlor ethylene showed it to have no measurable effect on the infants.

The Phenothiazine Derivatives

In recent years the phenothiazine drugs have been extensively used in the management of the obstetrical patient. When given in

an episiotomy under local analgesia with light general anaesthesia during delivery of the arms and head is satisfactory. In extraction great relaxation is required and deep ether is recommended.⁶⁹

Infant Resuscitation

A useful classification of asphyxia neonatorum recognizes three stages.⁵⁶ First is the stage of depression when the baby does not breathe well, has a tendency to get attacks of cyanosis, but in spite of all this is rousable. The respiratory centre, circulation, muscle tone and reflexes are all depressed from hypoxia but are capable of being stimulated to normal activity. Then comes the spastic stage when breathing is irregular and gasping and cyanosis is the rule; there is however some reflex response to aspiration of mucus from the larynx and pharynx. Lastly comes the stage of flaccidity when the child is blue or white and respiration is either in abeyance or occurs only at infrequent intervals. No reflex response is present and circulatory failure is progressive.

In a study of the human foetus in prolonged asphyxia⁵⁷ it has been shown that there is a steady decline in blood pressure and heart rate while the cardiac conduction time is increased. The blood pressure drops further during gasping respiration. Oxygenated heparinized blood injected into the umbilical vein causes a rapid and sharp rise in the heart rate and the blood pressure, and it seems to reach the left ventricle and the coronary arteries very rapidly. In the gravest cases, the best results appear to follow injection of the heparinized oxygenated blood into the foetal umbilical artery. Infusion of citrated blood and of noradrenaline are apparently harmful.

Asphyxia neonatorum is a chemical condition associated with a decrease in oxygen content and oxygen saturation, a rise in carbon dioxide tension and sometimes a fall in carbon dioxide content as a result of its displacement from base by the large increase in the amount of lactic acid present in the blood. The pH of the blood is very low.⁷⁰

It has always been understood that high positive pressure applied from above to the bronchial tree of the atelectatic infant is not very successful in opening up the alveoli but the author of a recent paper⁵⁸ gives good experimental and clinical evidence for holding the opposite view. He believes that it is not only possible but also beneficial to deliver a pressure necessary to expand the unexpanded lung of the newborn and has used for this purpose the Goddard-Bennett-Lovelace infant hand resuscitator with mask. The resuscitation routine is to establish a good foetal airway, promote adequate

The successful management of a case of post partum hæmorrhage with irreversible shock by induced hypothermia is the subject of an interesting report⁵³ The patient bled heavily on several occasions and as hysterectomy was considered necessary the operation was undertaken after the intravenous injection of chlorpromazine, promethazine pethidine thiopentone, and suxamethonium with gas and oxygen given through an endotracheal tube The rectal temperature was taken down to 35° C by means of surface cooling with icebags, and the blood pressure was supported by a noradrenaline drip Spontaneous re warming was not accompanied by stabilization of the vasomotor system so that additional infusion of phenothiazine drugs and surface cooling were required The authors of the report emphasize that every effort must be made to maintain the circulating blood volume at an adequate level before the lytic cocktail containing chlorpromazine is injected, because of the danger of causing peripheral vaso-dilatation when the circulating volume is deficient

Hypofibrinogenæmia

The occurrence of a blood coagulation defect in certain obstetrical emergencies is now well recognized and this has probably accounted for some cases of death due to hæmorrhage when good transfusion treatment was carried out It is probable that an acquired lack of fibrinogen is responsible for the coagulation defect⁵⁴ and this may occur in cases of accidental ante partum hæmorrhage retention of a dead foetus in utero and hydatiform mole Hypofibrinogenæmia can be diagnosed by a simple thrombin coagulation test before clinical signs have shown themselves so that patients who have a Schneider titre of 1/100 or less should be treated as potential victims of a hæmorrhagic diathesis If hæmorrhage occurs the blood fibrinogen level must be restored and this may be done by giving pure fibrinogen or, more easily by the intravenous infusion of triple or quadruple strength plasma One pint of the latter provides about 4.4 g of fibrinogen.⁵⁵ In addition blood must be given to replace any lost. The use of plasma expanders like polyvidone and dextran is not desirable in these cases as such agents do not restore the coagulability of the blood

Breech Delivery

Anæsthesia for breech delivery may present some problems In breech presentation the infant may be delivered in one of two ways as assisted breech delivery or as a breech extraction In the former

transparent plastic cylinder into which the child is put, head and body. This is then closed and oxygen and air are introduced under positive pressure at suitable intervals and at suitable pressures. It is provided with heat and humidity regulation and is said to give useful help in the management of this problem.

Obstetricians are not yet all agreed that endotracheal intubation is justified in the treatment of asphyxia neonatorum, but if the technique is employed it must be done carefully and correctly. The commonest mistake is to lower the head over the edge of the table to hyperextend it, but this is incorrect.⁶⁶ The child should be flat with its occiput on the table. The tip of the infant laryngoscope is then inserted to the glottis and lifted vertically when the cords will come into view and a number 00 Magill endotracheal tube on a rigid stylet can be placed in the trachea. In the absence of anyone skilled in direct laryngoscopy, good results can be obtained by the gentle insufflation of oxygen through a small double lumen catheter into the stomach.⁶⁷ The second lumen prevents gastric distension.

Nalorphine has achieved some success in the prevention and treatment of respiratory depression due to morphine or one of its congeners in the newborn.^{68, 69} The drug can be given intravenously in 10-mg doses to the mother just before delivery if she has recently had a narcotic of the opium or pethidine group and this may enable patients to be premedicated with morphine before a planned Cæsarean section. It is probably better to give the drug in 0.1 to 0.5 mg doses into the umbilical vein of the child if and when it is indicated. In such cases prompt improvement in respiration usually follows. As the nalorphine is in its own right a narcotic and a depressant it must only be used in those babies depressed by an opiate pethidine meperidine domoran etc. The stimulating effect of the drug on respiration usually subsides before the depressant action of the morphine so the infant may become depressed again.

The new drug amiphenazole (daptazole) would seem to have a definite place in the treatment of neonatal asphyxia due to morphine or to one of its congeners. After attention to the airway has been gently and thoroughly carried out 3 mg of the drug are injected dissolved in 1 ml of saline into the infant's umbilical vein and milked towards the navel. Gasping respirations appear in half to one minute and soon give place to normal respiration.⁷⁰ Amiphenazole can also be given intravenously to the mother who has received morphine just before the delivery of the child as it does not interfere with or prevent the analgesic properties of this and similar sedatives. To both mother and child it is said to be harmless even in large doses.

drainage of the upper respiratory tract by posture and suction expand the lungs by intermittent positive pressure, administer oxygen and such stimulants as nalorphine or caffeine sodium benzoate if they are indicated maintain temperature and humidity and stimulate the skin. Using the hand resuscitator first at a pressure of 50–60 cm of water twelve to twenty four impulses are given each lasting 0.2 to 0.3 of a second allowing an interval of 0.4 to 0.8 of a second for expiration. Suction is then applied and a series of inflations at a slightly lower pressure follow. Intubation is seldom required and oxygen blown into the stomach is readily expelled by manual epigastric pressure. X ray studies of infants so treated have failed to show any evidence of pneumothorax or of other damage. Failure to respond to this vigorous treatment in twenty minutes is said to be evidence in nearly every case of intracranial damage or of congenital defects. The author concludes that patchy aeration of the atelectatic neonatal lung is achieved with a positive pressure of 30 cm of water and uniform expansion at pressures of 50–60 cm of water in the closed chest. Negative pressure is not required. Following the initial expansion pressure must be reduced. Further experimentation with this interesting technique would seem to be desirable.

Another method of artificial respiration in the asphyxiated infant is the application of Eve's rocking technique⁵⁹ by the use of an automatic electrically operated rocking bassinet.⁶⁰ Oxygen is added to the air in the chamber in which the child lies which has an open top. In 4000 consecutive cases reported⁵⁹ no other form of resuscitation was used and the results were excellent. An experimental high tension oxygen box incorporating cooling and humidifying devices has been described.⁶¹ In it the minimal concentration of oxygen is 92 per cent and carbon dioxide concentration does not exceed 1 per cent and so it is stated to be more efficient than the standard oxygen tent for use in the treatment of severe anoxia in babies. It is not intended as an incubator for premature babies. A very simple device for oxygen therapy in asphyxia neonatorum consists of a glass funnel 3–4 in. in diameter with a length of rubber tubing fitting snugly and protruding into the bell.⁶² The funnel is applied to the baby's face so that the rubber tube is near its upper lip. A flow of 3 or 4 litres of oxygen a minute is directed through the rubber tube and simultaneously stimulates nerve endings of the trigeminal nerve and supplies oxygen in fairly high concentration for the resulting inspiration. A positive pressure air lock has been constructed for the resuscitation of the newborn.⁶³ It consists of a

- 25 BERNSTEIN, U B MEYER A E and HAYMAN H B 1954 *J Obstet Gynec Brit Emp* 61 683 (Oct)
- 26 KANE W M 1953 *Amer J Obstet Gynec* 65 1020 (May)
- 27 ROBERTS H and WRIGLEY F W 1953 *J Obstet Gynec Brit Emp* 60 538 (Aug)
- 28 POWELL, P E. (jun) and SAVAGE J E 1953 *Obstet et Gynec* 2 658 (Dec)
- 29 ENRICH J P (jun) 1955 *Amer J Obstet Gynec* 69 124 (Jan)
- 30 ROSENFELD S S *et al* 1954 *Amer J Obstet Gynec* 67 1067 (May)
- 31 CONDEN A M 1951 *J med Ass Alabama* 21 53 (Sept)
- 32 HINGSON R A and HELLMAN L M 1954 *Amer J Obstet Gynec* 68 262 (July)
- 33 MORDHAVAN P and BHASKER K 1955 *J Obstet Gynec Brit Emp* 62 589 (Aug)
- 34 JONES G R *et al* 1952 *Sth med J* 43 34 (Jan)
- 35 BRYCE SMITH R and WILLIAMS E O 1955 *Lancet* 1 1241 (June 18)
- 36 LESSER M 1954 *Brit med J* 2 79 (July 10)
- 37 PITTINGER C B and MORRIS L E 1955 *Anaesth & Analges* 34 107 (March-April)
- 38 PITTINGER C B and MORRIS L E 1953 *Anesthesiology* 14 238
- 39 ELLEKER A R 1950 *Brit med J* 2 398
- 40 LITTLE D M *et al* 1953 *Anaesth & Analges* 22 171 (May-June)
- 41 THESLEFF E 1952 *Acta physiol scand* 27 Suppl 99
- 42 McNAB J A 1955 *Canad med Ass J* 72 681 (May 1)
- 43 APOAR V and PAPPER E M 1952 *Anaesth & Analges* 31 309
- 44 McKECHNIE F B and CONVERSE J G 1955 *Amer J Obstet Gynec* 70 637 (Sept)
- 45 DYBING O and STROMORKEN H 1952 *Acta pharmac et toxicol* 8 271
- 46 TAYLOR E S *et al* 1954 *Amer J Obstet Gynec* 69 348 (Feb)
- 47 BENARON H H W *et al* 1955 *Amer J Obstet Gynec* 69 776 (April)
- 48 DAVIES J I 1955 *Canad Anaesth Soc J* 2 327 (Oct)
- 49 KARP M *et al* 1955 *Amer J Obstet Gynec* 69 780 (April)
- 50 SAVAGE D 1955 *Brit J Anaesth* 27 346 (July)
- 51 CARROLL, J J and HUDSON P W 1955 *Canad Anaesth Soc J* 2 340 (Oct)
- 52 PEZZOLI C 1955 *J Amer med Ass* 158 87 (May 7)
- 53 MURRAY D H and BRUCE D F 1955 *Lancet* 2 699 (Oct 1)
- 54 BARRY A P GEOGHEGAN F and SHEA S M 1955 *Brit med J* 2 287 (July 30)
- 55 SCOTT J S 1955 *Brit med J* 2 290 (July 30)
- 56 FLAGG P J 1944 *The Art of Resuscitation* New York Rheinhold Publishing Corp
- 57 ENKORNING G and WESTIN H 1954 *Acta physiol scand* 31 358
- 58 GODDARD R F 1955 *Anaesth & Analges* 34 1 (Jan)
- 59 MILLER R S *et al* 1955 *Amer J Obstet Gynec* 70 1087 (Nov)
- 60 MILLER R S and DAVIES J 1946 *Amer J Obstet Gynec* 52 508
- 61 MORRISON H 1955 *Lancet* 2 752 (Oct 8)
- 62 SKERREN Y 1954 *J Obstet Gynec Brit Emp* 61 447 (Aug)
- 63 ZELENIK J B and PRYSTOWSKY H 1952 *Amer J Obstet Gynec* 64 1316
- 64 MANN J 1954 *Anaesth & Analges* 33 289 (Sept-Oct)
- 65 GREEN H A 1955 *Amer J Obstet Gynec* 70 618 (Sept)
- 66 CHALMERS J A and THORNBERRY C J 1954 *J Obstet Gynec Brit Emp* 61 244 (April)
- 67 AKERREN Y and FURSTENBERG M 1950 *J Obstet Gynec Brit Emp* 57 705
- 68 PENTFIELD WILDER 1954 *Anaesth & Analges* 33, 145 (May-June)

Amiphenazole given to the mother in 30 mg doses intramuscularly reduces the respiratory depressant effect of morphine on the fœtus without greatly reducing its analgesic action on the mother. With this new drug to hand it would seem that morphine can now be used much more freely in hospital obstetric practice than was formerly desirable.⁷⁸

Oxygen Therapy in Premature Babies

Premature babies must not receive pure oxygen for any length of time. If they are cyanosed they may be given for a minimal period 40 per cent oxygen. If this rule is not followed retrolental fibroplasia may result.⁷¹

The Dangers of Childbirth

In considering this obstetrical and anæsthetic problem it is interesting to remember that Wilder Penfield believes⁶⁸ that well over half the patients on whom he operates for focal epilepsy would never have had a seizure had it not been for brief anoxia or improper head compression during childbirth.

References

- 1 GIBBERD G F 1955 *Lancet* 1 901 (April 30)
- 2 PARKER R B 1954 *Brit med J* 2 65
- 3 MENDELSON C L 1946 *Amer J Obstet Gynec* 52 191
- 4 HAUSSMAN W and LUNT H L 1955 *J Obstet Gynec Brit Emp* 62 509 (Aug)
- 5 LOCK F R and GREISS F C 1955 *Amer J Obstet Gynec* 70 861 (Oct)
- 6 WOOLSEY E L 1955 *Amer J Obstet Gynec* 69 128 (Jan)
- 7 HARRIS J R 1954 *Amer J Obstet Gynec* 68 969 (Oct)
- 8 GATE J M and DUTTON W A W 1955 *Brit med J* 2 99 (July 9)
- 9 KLINK E 1953 *Obstet et Gynec* 1 137
- 10 BEST D 1954 *Canad Anæsth Soc J* 1 10 (July)
- 11 EGERTON C D 1954 *Amer J Obstet Gynec* 62 1098 (Oct)
- 12 THORNE T C 1954 *Proc R Soc Med* 47 301 (May)
- 13 THORNE T C 1956 personal communication
- 14 COPE R W 1954 *Anæsthesia* 9 249 (Oct)
- 15 MACINTOSH R R 1954 *Proc R Soc Med* 47 312 (May)
- 16 MEHL L H 1954 *Amer J Obstet Gynec* 68 1105 (Oct)
- 17 FLOWERS C E 1954 *Anæsthesia* 9 146 (July)
- 18 GORDON C A, ANSBRO F P and GORDON H E 1955 *Amer J Obstet Gynec* 69 1203 (June)
- 19 ANSBRO F P *et al* 1952 *N Y State J Med* 52 1901 (Aug 1)
- 20 ARTHUR H R and JOHNSON G T 1952 *J Obstet Gynec Brit Emp* 59 372 (June)
- 21 JOHNSON G TREVOR 1954 *Brit med J* 1 627 (March 13)
- 22 HINGSON R A and EDWARDS W B 1943 *J Amer med Ass* 121 225
- 23 JEWELL T C 1954 *Amer J Obstet Gynec* 67 273 (Feb)
- 24 THOMAS B E and GIBSON J 1953 *J Obstet Gynec Brit Emp* 60 378 (June)

CHAPTER 19

COMPLICATIONS OF ANÆSTHESIA

Acute adrenocortical deficiency—Pulmonary complications—Laryngeal complications—Vomiting—Surgical emphysema—Nerve palsies—Blindness—Toxic effects of carbon dioxide accumulation—Perforation of ear drums—Explosion—Pneumo thorax—Cerebral hypoxia

THE complications of anæsthesia are unfortunately rather numerous so that two books published in recent months are devoted solely to their consideration. But when set against the total number of anæsthetics administered, the picture takes on a less gloomy appearance. The complications which are considered in this chapter include only those which have been brought to the attention of anæsthetists in recent years either for the first time or for purposes of re-emphasis.

ACUTE ADRENOCORTICAL DEFICIENCY

If a patient is treated with cortisone for any length of time his own adrenal cortex may undergo atrophy through disuse and may not regain its normal activity for a considerable time after cessation of cortisone treatment. If now such a patient is to be subjected to surgical operation and anæsthesia, a true stress situation his cortex may not be in a fit state to deal with it. Any patient who has received a substantial amount of cortisone within three or six months should therefore receive prophylactic cortisone therapy before being submitted to surgery. An intense suprarenal stimulation occurs shortly after the beginning of the operation and persists for one or two days. While acute post operative deficiency is rare a rise in its incidence can be expected because of the increasing use of cortisone. Patients should be asked before operation if they have been treated with cortisone recently. It may well have been given to those suffering from rheumatoid arthritis, ulcerative colitis, lupus erythematosus, peri arteritis nodosa or status asthmaticus.⁵ Various tests of the reaction of the suprarenal cortex to stress have been described,¹ while the eosinophil count is valuable in the immediate post operative period.⁶ In the presence of shock a high count is indicative of suprarenal cortical deficiency and it may well be that certain unexplained deaths following relatively minor operations have been due to this cause.^{2, 4, 7} It has also been suggested that

- 69 LAW R G and RANSOM S., 1954 *Brit med J* 1 562 (March 6)
- 70 TOYELL, R M., BANNISTER W K., and LITTLE, D M., 1965 *J Amer med. Ass* 159 1337 (Dec. 3)
- 71 AUSTON N., *et al.*, 1953 *Brit J Ophthal.*, 37 543
- 72 CRAWFORD J S., 1956 *Brit J Anæsth* 28 146 and 28 201 (April)
- 73 WYLIE W D., 1956 *Lancet* 1 840
- 74 WYLIE, W D., 1955 *Proc Roy Soc Med.*, 48 1059 (December)
- 75 ROCKER L., 1956 *Brit med J.*, 2 78 (July 14)
- 76 CRAWFORD J S and GARDINER J E. 1956 *Brit J Anæsth* 28 1-6 (April)
- 77 HOLMES, J M. 1956 *Lancet* 2, 334 (Aug. 18)
- 78 HOLMES, J M., 1956 *Lancet* 2 765 (Oct 13)

and on their return to the ward are similarly dealt with as soon as they are able to be moved about. After operation the treatment is continued until no more sputum can be coughed up.

A method of separating those patients who are likely to develop chest complications after operation from the remainder, is to ask each patient to cough deeply before induction. The wet cases will need careful aspiration and physical therapy.⁸ For those patients who, having sputum, are unable to cough it up after operation, the intravenous injection of 2 ml. of paraldehyde will often cause an explosive cough with a clearance of the bronchial tree.⁸ In bronchiectatic patients the treatment must be used carefully as it may result in flooding of the bronchial tree with pus. The severe coughing and straining which accompanies any successful stir up treatment has been known to lead to subarachnoid hæmorrhage.⁹ Temporary paralysis of the diaphragm may be seen occasionally after laparotomy¹⁰ and is likely to be mistaken for atelectasis. Aspiration and bronchoscopy are not the treatments of choice in this condition, the possibility of which should be borne in mind. In order to ease coughing and to increase the amount of sputum expectorated, use has been made of *alevaire*, a detergent in sterile alkaline solution which can be used alone or as a vehicle for antibiotics and bronchodilators.¹¹ It lowers the surface tension and liquefies mucopurulent secretion. It should be nebulized by compressed oxygen or air so that a fine mist is inhaled into the most distal alveoli. One to four periods of treatment, each lasting twenty to ninety minutes, are given each day and deep breathing and coughing exercises follow the inhalation.

Spontaneous atelectasis coming on during the course of an operation would seem to be a genuine occurrence¹² and has been observed during a thoracotomy. It may be due to increased vagal activity,¹³ a fact supported by the success which has attended the injection of a local analgesic solution into the vagus below the origin of the recurrent laryngeal nerve. Tension pneumothorax can complicate undetected fracture of the ribs causing trauma to the underlying lung.¹³ It is important that X rays should be taken when a chest injury is even remotely possible. If a lung injury is suspected regional analgesia might well be suitable but pneumothorax may develop even with spontaneous respiration. Aspiration of air or gas from the thorax should be done before induction of anaesthesia. If the condition is recognized before operation then the insertion of an intercostal catheter connected to an underwater seal, with controlled breathing would be reasonable.

some cases of so-called status lymphaticus may be due to this mechanism³

Prophylactic therapy which should also be given to patients before operation who have Addison's disease, pituitary insufficiency suprarenal cortical tumours and those about to undergo bilateral adrenalectomy might consist of 200 mg. of cortisone intramuscularly forty-eight twenty four and two hours before operation. This is continued in a smaller dosage for three or four days or until their condition warrants withdrawal⁴. All such patients should be followed carefully in the immediate post-operative period for fear of the development of acute insufficiency and frequent estimations of the blood pressure temperature and pulse rate should be made. They tolerate badly prolonged fasting morphine and barbiturates and large volumes of intravenous glucose. Acute insufficiency usually shows itself in the first twenty four hours after operation and appears as acute circulatory collapse hypotension and tachycardia perhaps with fever and unconsciousness. The treatment is intravenous saline intravenous hydrocortisone 100 mg given as a drip and if necessary noradrenaline.

CHEST COMPLICATIONS

The prevention of post-operative chest morbidity is the subject of a thoughtful paper by Palmer and Sellick. Their method is based on the hypothesis that if factors which favour the retention of sputum in patients after operation can be overcome atelectasis should seldom occur. It consists of postural drainage with vibratory and clapping percussion following the inhalation of isoprenaline. This drug is a powerful bronchodilator and vasoconstrictor of the bronchial mucosa. It is given to overcome the spasm and congestion of the bronchi which are thought to be responsible for the rhonchi often heard in patients who develop atelectasis. Pre-operative treatment consists of the oral inhalation of 1 ml. of 1 per cent isoprenaline sulphate (neopamine solution No. 1 alendrine, isupren, neodrenal) by means of a hand inhaler thrice daily followed by fifteen to twenty minutes postural drainage with the foot of the patient's bed raised, and percussion to the basal regions in expiration. It is done in the prone and in both lateral positions. If there is marked bronchitis two weeks of treatment along these lines is prescribed and in less severe cases one week. By adopting this method the incidence of atelectasis after operation was reduced from 43 per cent to 9 per cent. compared with a similar control series.⁵ The patients are clapped and given the treatment just before the premedication is injected.

arytenoid and it has been asserted¹⁶ that careful inspection will reveal signs and symptoms of laryngeal pathology in 50 per cent of cases after endotracheal intubation. The prognosis of cases of minor trauma is good given early diagnosis and complete vocal rest. The granuloma once developed should be removed under direct laryngoscopy and the results of this small operation are usually very good. It is interesting to realize that the autonomic movements of the cords are not abolished during light anaesthesia¹⁵ so that if a tube is in place the tips of the vocal processes are rubbed to and fro in close contact with it, so that in fact the lesion may not be due to trauma produced by the insertion or withdrawal of the tube at all.¹⁸

Acute oedematous stenosis is more usual in children than in adults because of the loose areolar tissue in the child's subglottic region and the small lumen which is occluded by very little swelling. When the acute oedema is caused by a tube the introduction of another tube is contra-indicated and a low tracheotomy should be done. Symptoms of laryngeal obstruction include indrawing of any one or more of the following locations—the suprasternal notch, the intercostal spaces, the areas round the clavicles or the epigastrium. There is an ashy grey pallor, cyanosis being a dangerously late sign, and choking, gagging and restlessness due to air hunger. The child may wake in terror and may fear the approach of suffocation. Laryngeal obstruction must of course never be treated by sedatives, it demands immediate tracheotomy. Avoidance of physical and chemical trauma is of the greatest importance in prevention of this condition.

In another series¹⁷ twelve adult cases of laryngeal lesions following intubation were reported, the majority of them in women. In some cases symptoms were absent throughout while in others, complaints arose long after the operation. Hoarseness, intermittent aphonia, continuous dyspnoea, pain, haemoptysis and a feeling of a foreign body in the throat have all been complained of. The pathology appears to be abrasion leading to infection, ulceration, granuloma formation and organization to a firm nodule. Even an easy intubation and extubation with immediate post-operative recovery has been known to cause rapidly fatal asphyxia on the second day.¹⁸

Occasionally an anaesthetist will experience difficulty in removing a tube which gave no trouble on its insertion. This of course is more common when a cuffed tube is used. If muscle spasm is the cause of the difficulty the injection of a small dose of a relaxant will remove it.¹⁹ In another case known to one of the authors the cuffed tube held fast in spite of relaxants, carbon dioxide, rotation, etc. and eventually was withdrawn only after the application of a frightening

Surgical emphysema may be seen during the administration of an anæsthetic or its onset may be delayed until the patient is back in bed. One theory of the mechanism of its causation¹⁴ is that over inflation of the alveoli by hyperventilation causes alveolar distension and rupture resulting in pulmonary interstitial emphysema. The gas then travels with the vessels to the lung hilum causing mediastinal emphysema and this may spread up into the neck, down with the œsophagus and aorta into the retroperitoneal tissues or it may rupture into one or both pleural cavities resulting in tension pneumothorax. If there is a wound round about the base of the neck air may be sucked into the mediastinum and there imprisoned when the wound is closed. This state of affairs is most likely to take place if the anæsthetist has allowed respiratory obstruction to persist with its hyperpnœa and increased intrathoracic negative pressure. Injury to the mucosa of the upper respiratory tract together with intermittent positive pressure respiration may also result in surgical emphysema, while it has been observed to occur spontaneously during normal labour. If surgical emphysema develops in the subcutaneous tissues the onset of pneumothorax must be watched for and if this is shown to be present it should be treated by aspiration in the third intercostal space in the mid clavicular line and a water seal drain established. If the abdomen becomes distended the flanks should be aspirated with a syringe and large needle. Should respiratory difficulty not due to obstruction develop then a collar incision can be made, the mediastinum opened up by blunt dissection and the imprisoned gas let out. Emphysema confined to the subcutaneous tissues is comparatively harmless but should be aspirated repeatedly.

LARYNGEAL COMPLICATIONS

Chevalier Jackson¹⁵ has reviewed in a masterly way the laryngeal complications of endotracheal intubation. He pays particular attention to contact ulcer granuloma and acute laryngeal œdema requiring tracheotomy. The former lesion not infrequently occurs without intubation or other major trauma and is due to the wear and tear of the vocal cords in speaking and coughing. The hammering impact of the cartilaginous vocal process of one arytenoid against the opposite arytenoid is one such cause and it leads to injury of the mucoperichondrium of one or both sides and the establishment of a contact ulcer which may proceed to granuloma formation. Abuse of the voice is a second cause while persistent cough is a third. The usual site of the granuloma is the tip of the vocal process of the

N benzhydryl N methyl piperazine dihydrochloride It has an antihistamine effect and blocks parasympathetic ganglia. It has been found experimentally to reduce the incidence of vomiting in dogs who have received apomorphine and in a controlled series in man reduced vomiting by 23.9 per cent.³ It is said to cause no side effects. It may be injected hypodermically half an hour before the end of the operation and repeated once or twice after operation, the dose being 50 mg.³

The prevention of the aspiration of stomach contents during induction of anaesthesia is one of the anaesthetist's ever present problems. It can be dealt with in several ways, no one of which is universally applicable or absolutely safe. One new method is to induce anaesthesia in the reverse Trendelenburg position to prevent regurgitation with cyclopropane and oxygen, 50 per cent of each and then suxamethonium is given intravenously and a cuffed endotracheal tube smoothly and rapidly inserted and its cuff inflated.³⁰ The cyclopropane is less likely to cause a serious fall in blood pressure than an intravenous barbiturate and induction is almost as rapid as by the intravenous method. It has been reported³¹ that if a Miller Abbott tube is introduced into the stomach and its cuff inflated with 30-60 ml of air and if it is then withdrawn until the balloon is tightly impacted against the oesophageal orifice nothing will be able to leak past it into the oesophagus so that the patient will be protected against aspiration. (See also under Intestinal Obstruction.)

COMPLICATIONS INVOLVING THE NERVOUS SYSTEM

The opponents of spinal analgesia are always willing to blame this method for any case of third nerve palsy which arises during the post operative period, providing of course that this block has been used. Third nerve palsy can however show itself after operations conducted with the patient under general anaesthesia and a new case of this has recently been reported.³² Horner's syndrome is a frequent minor complication of brachial plexus block and is expected after stellate ganglion block but its presence has been reported after block of the lumbar sympathetic ganglia.³³ Supra-orbital neuropraxia as a complication of anaesthesia should be constantly borne in mind. The patient afflicted with this disorder will complain of pain in the eye, headache, photophobia and numbness over one side of the forehead and scalp with oedema of the upper lid and chemosis. It is due to pressure on the supraorbital nerve of a badly fitting mask or of a rubber or metal tube. It is

degree of force Three days later signs of laryngeal obstruction were in evidence so that tracheotomy became necessary The tracheotomy tube was removed at the end of a week and the patient an old lady of seventy, made a perfect recovery

Obstruction of the larynx during anæsthesia is usually due to spasm of the cords but two other types have been observed ²¹ In one there is spasm of the false cords and aryepiglottic folds In the other a condition has been observed where adduction of the true cords coincides with inspiration and this leads to obstruction It would seem to be a true inco-ordination of the cord mechanism Unusual stenosis of the adult trachea has been described ²² It caused failure to introduce an endotracheal tube of suitable size more than a short distance beyond the cords and proved to be due to an unusual narrowing of the trachea without any evidence of disease Impaction of the epiglottis during anæsthesia has again been described ²³ It seems that three factors must be present for this to happen first, an anatomical abnormality the overhanging epiglottis then muscular relaxation and finally the gravity effect consequent on the supine position (see also under Endotracheal Anæsthesia)

VOMITING

One of the more useful pharmacological effects of chlorpromazine is its ability to reduce the incidence of post operative vomiting ³ It has been shown to be a valuable drug in the pre- and post operative periods when given by mouth to patients undergoing major thoracic procedures while for abdominal cases 300 mg rectal suppositories are under trial ²⁴ A suitable schedule for oral dosage is 25 mg given the night before operation and a similar amount before operation after operation and three times during the first post operative day It has been claimed ⁵ that the oral administration of 50 mg before operation reduces the post operative vomiting rate by 50 per cent Dimenhydrinate (dramamine) has also achieved a useful reputation as an effective means of prevention of post operative vomiting When given intramuscularly in 50 mg doses before operation after operation and then in the immediate post operative period for four doses it has been shown to reduce vomiting by 50 per cent ²⁵ On the other hand when given by mouth dimenhydrinate is not very effective ²⁷ Promethazine when given parenterally significantly reduced the vomiting in general surgical cases and in obstetrical patients without in the latter causing any foetal depression ²⁷ Yet another new drug used for the relief of this most uncomfortable condition is cyclizine (marzine, marazine) which chemically is

arises in old people its cause must be sought and the vicious circle confusion hypnotics more confusion must not be allowed to occur. Some anaesthetists feel that in this article the picture has been overdrawn but it is a useful reminder that the stress of operation and anaesthesia must not be lightly inflicted on patients who are handicapped either by old age or anything else.

Symptoms due to haemolysis have been reported during and after the operation of transurethral prostatectomy⁴³ and have been found to be due to the presence in the blood stream of some of the fluid used for bladder irrigation. The patient complains of lassitude and chills if he is under spinal analgesia and shows bronchospasm, clammy skin, a slowing of the pulse and a rise in blood pressure. It is recommended that isotonic solution should be used for irrigation in these cases.

In recent years, a good deal of attention has been devoted to the toxic effects of carbon dioxide accumulation³⁶ and a considerable amount of evidence has come to light pointing to the grave harm that may result from this form of physiological trespass. The use of central respiratory depressants and relaxants will favour this accumulation which may have bad or even lethal effects on both the heart and circulation. It may also cause further respiratory depression which persists even after the body has got rid of other depressants and relaxants³⁷. Artificial hyperventilation would be required before the respiratory centre recovered from this carbon dioxide induced depression. The toxic accumulation of carbon dioxide leading to respiratory depression may follow a period of respiratory arrest or hypoventilation and may well be a cause of death in the immediate post operating period.

EXPLOSIONS DURING OPERATIONS

The use of conducting rubber is becoming widespread in our operating theatres but the consistency of this material tends to be hard and to lack elasticity. It is to be hoped that the manufacturers will overcome their present difficulties before very long. That combustible and explosive material is not always brought into the theatre by the anaesthetist is shown in an article on the generation of flammable gases in the patient's alimentary canal⁴⁴ both in the lower gut and also in the stomach as a result of fermentation when emptying is delayed. Oxygen swallowed or blown into the stomach during intermittent positive pressure respiration may add to the risk of explosion of gases already in the alimentary canal. To prevent explosion when the diathermy is used through a proctoscope or

wise to protect this area with either sorbo rubber or gamgee tissue³¹ Two cases of unilateral blindness following hypotensive anæsthesia have been described³² In each case there was in addition to a rather low blood pressure some pressure on the eyeball The retinal artery is an end artery and blood entering the eye through it does so against the intraocular pressure If the blood pressure is reduced and the intraocular pressure increased, disaster may result

Hypertension sometimes occurs during anæsthesia in patients with transection of the spinal cord at the fifth dorsal segment or above³³ Massive effects on the autonomic nervous system occur when such stimuli as distension of the bladder or rectum arise³⁴ The part of the cord below the lesion not only recovers its reflex function but becomes highly excitable so that various stimuli may lead to hypertension The ideal treatment of this alteration in blood pressure is of course to remove the stimulus causing it but the trigger mechanism is not always apparent so that in these cases a ganglion blocking agent such as hexamethonium may be useful Extradural or subarachnoid block also prevents the reflex³⁵ A rapidly fatal hyperpyrexia after the administration of thiopentone, gas oxygen and trichlorethylene to a fit young girl of thirteen has been reported⁴⁰ Another unusual complication of anæsthesia which has been described is perforation of the eardrum⁴¹ Intermittent positive pressure respiration was used and presumably this compressed the air and gases in the mouth and pharynx so that the pressure in the eustachian tubes was increased sufficiently to cause rupture of a thin atrophic drum In the case reported antibiotics and cleanliness comprised the treatment and the patient made a good recovery

An article published in the medical press on the adverse cerebral effects of anæsthesia in old people⁴ has caused a great deal of discussion and some heart searching The author, a physician particularly interested in old people made personal observations on his own patients before and after operation and came to the conclusion that dementia in its lesser degrees of severity is not rare after operation and anæsthesia while in addition severe dementia is described in eighteen of his patients As a result of this investigation he feels that operations on the elderly should be confined to necessary cases and that sedative drugs and narcotics should be most carefully administered During and after operation the blood pressure pulse rate hæmoglobin and oxygen level of the blood should be maintained at optimal levels while hypotensive anæsthesia is absolutely contra-indicated in this type of patient When post operative confusion

- 5 SPENCE SALES D and MEHTA N 1955 *Med Illus* 9 639 (Oct)
- 6 ROOT B., 1955 *Anæsth & Analges* 34 78 (March-April)
- 7 PALMER, K. N. V. and SELICK, B. A. 1953 *Lancet* 1 164
- 8 GREENE, H. A. and BERKOWITZ, S. 1953 *Anesthesiology* 14 166 (March)
- 9 MOORE, D. C. 1954 *Anesthesiology* 15 211 (March)
- 10 GAGE, A. A. and CHARDACK, W. M. 1953 *Amer J Surg* 85 232 (Feb)
- 11 RUBIN E. J. JAMISON W. and RAO K. V. S. 1955 *Anesthesiology* 16 801 (Sept)
- 12 GRIGOR, K. C. 1954 *Anæsthesia* 9 185 (July)
- 13 FAIRLEY H. H. 1955 *Anæsthesia* 10 375 (Oct)
- 14 SPENCE, M. 1955 *Anæsthesia* 10 50 (Jan)
- 15 JACKSON CHEVALIER 1953 *Anesthesiology* 14 425 (Sept)
- 16 MALONEY W. H. 1954 *Laryngoscope* 61 861 (Oct)
- 17 YOUNG N. and STEWART S. 1953 *Brit J Anæsth* 25 32 (Jan)
- 18 STOUT R. J. and THOMAS CLEMENT 1954 *Brit J Anæsth* 26 35 (Jan)
- 19 CULLINGFORD D. W. J. 1954 *Brit J Anæsth* 26 187 (May)
- 20 CAIGER, G. H. and SICHEL, D. A. S. 1954 *Anæsthesia* 9 177 (July)
- 21 STEWART J. and PINKERTON H. H. 1955 *Brit J Anæsth* 27 492 (Oct)
- 22 POKRZYWNICKI S. 1953 *Anæsthesia* 8 47 (Jan)
- 23 DUNDEE, J. W. 1954 *Brit J Anæsth* 26 357 (Sept)
- 24 BOULTON T. H. 1955 *Anæsthesia* 10 233 (July)
- 25 ALBERT S. N. and COAKLEY C. S. 1954 *Anæsth & Analges* 33 285 (July-Aug)
- 26 MOORE, D. C. *et al.* 1955 *J Amer med Ass* 159 1342 (Dec 3)
- 27 GORDON R. A. *et al.* 1954 *Canad Anæsth Soc J* 2 95 (Oct)
- 28 DENT S. J. RAMACHANDRA V. and STEPHEN C. R. 1955 *Anesthesiology* 16 564 (July)
- 29 MARCUS P. S. and SHEEHAN J. C. 1955 *Anesthesiology* 16 423 (May)
- 30 WYLIE, W. D. 1955 *Proc R Soc Med* 48 1089 (Dec)
- 31 FISHER, C. W. 1953 *Anesthesiology* 14 506 (Sept)
- 32 NORMAN J. E. 1955 *Anæsthesia* 10 87 (Jan)
- 33 EGBERT L. D. 1955 *Anesthesiology* 16 811 (Sept)
- 34 BARRON D. W. 1955 *Anæsthesia* 10 374 (Oct)
- 35 GILLAN J. G. 1953 *Canad med Ass J* 69 294 (Sept)
- 36 SCURR C. F. 1954 *Brit med J* 1 565
- 37 PASK E. A. 1955 *Anæsthesia* 10 4 (Jan)
- 38 GUTTMANN L. and WHITTERIDGE D. 1947 *Brain* 70 361 (Dec)
- 39 CILIBERTI, B. J. GOLDFEIN J. and ROVENSTINE E. A. 1954 *Anesthesiology* 15 273 (May)
- 40 BROWN R. C. 1954 *Brit med J* 2 1526
- 41 WHITTINGHAM J. K. R. 1954 *Brit med J* 2 970
- 42 BEDFORD P. D. 1955 *Lancet* 2 259 (Aug 6)
- 43 MINUCK, N. 1954 *Canad Anæsth Soc J* 1 59 (Oct)
- 44 GALLEY A. H. 1954 *Brit J Anæsth* 26 189 (May)
- 45 HUNTER, A. N. 1955 *Proc R Soc Med* 48 765 (Oct)
- 46 ARGENT D. E. and COPE D. H. F. 1956 *Brit med J* 1 593 (March 17)
- 47 LUNDY J. E. 1955 *Proc Mayo Clin* 30 No 2 446 (Oct 5)
- 48 CHURCHILL DAVIDSON H. C. 1956 *Proc World Congress of Anæsthesiologists* (1955) p 13 Burgess Minneapolis

sigmoidoscope, a stream of carbon dioxide should be directed into the lumen of the instrument, both to dilute and to blow away the flammable mixture of gases which might be present (See also Chapter 5)

PROBLEMS OF ANÆSTHESIA IN ARTIFICIAL PNEUMOTHORAX

Complications are apt to arise when ordinary anæsthetic techniques are employed in patients who have either an artificial pneumothorax or a pneumoperitoneum⁴⁵ The first sign of trouble may be a rising pulse rate an increase in the amount of venous bleeding tachypnœa and a falling blood pressure The cause of these troubles has been shown to arise from an increase of gaseous pressure in the chest which develops during and after anæsthesia⁴⁵ When nitrous oxide and oxygen are used for induction the tension of the nitrogen in the blood falls while that of nitrous oxide rises rapidly The nitrogen in the pneumothorax moves into the blood stream more slowly than the nitrous oxide diffuses into the chest owing to the difference in solubility of the two gases and there is thus a marked increase in the total gas content in the space and hence an increase in pressure It is this rise in intrathoracic pressure which causes increased venous pressure and cardio respiratory embarrassment and care must be taken to remove excess gas from air containing body cavities before or during general anæsthesia

CEREBRAL HYPOXIA

Quite independently of cardiac arrest episodes of acute hypoxia may result in cerebral œdema The clinical signs may be complex and may include coma convulsions hyperpyrexia restlessness and choreoathetosis If the condition is suspected it should be treated by the intravenous injection of 50 per cent sucrose Unlike glucose sucrose does not pass into the cerebrospinal fluid and raise its osmotic pressure It is excreted by the kidney causing a diuresis The initial injection should be 40 ml which can be repeated once or twice at intervals of 15 minutes It should be followed by 10 per cent dextran given at the rate of 50 ml per hour⁴⁶

References

- 1 BAYLISS R I S 1955 *Brit med J* 1 495
- 2 LUNDY J S 1953 *Anesthesiology* 14 376
- 3 BEARD A J W 1955 quoted by SPENCE SALES D *et al Med Illus* 9 639 (Oct)
- 4 SALASSER R M *et al* 1953 *J Amer med Ass* 155 1509 (Aug 15)

after effects. The psychical part of post operative vomiting, for example, which is so marked in certain nervous and hysterical types of patients can often be entirely eliminated by suitable suggestion made during the induction of anæsthesia with nitrous oxide and oxygen. It is even held by some authorities that a strong suggestion of muscular relaxation made during the induction period will minimize the dosage of anæsthetic subsequently necessary to obtain adequate relaxation.³ It need hardly be said that for suggestion to be effective an absolutely quiet induction room is essential (see later) and no interference with the patient can be permitted by well meaning nurses or others.⁴ As a matter of fact, a good anæsthetist does actually employ suggestion, whether he consciously realizes it or not. It has been aptly remarked⁵ that 'personality (in the anæsthetist) is non toxic and does not throw any strain on the (patient's) heart, liver or kidney, nor does it depress respiration. This is more than can be said of many types of premedication. Suggestion also plays a large part in operations performed under local analgesia only,⁶ and in certain countries the psycho-anæsthetist is a regular institution. For example Pitkin describes his auburn haired vamp who has the faculty of making a woman forget that she is in the operating room—and makes the men feel that the operation could go on for ever if she would only remain with them!'⁷ If pure local analgesia is to be used, the whole operating team must co-operate wholeheartedly. Even now it is not very uncommon for the surgeon to make his incision at the same time asking in a loud voice 'Do you feel that?' There is no doubt that local analgesia applied indiscriminately and with no attempt at suggestion does frequently result in psychic shock and actual insanity has occurred as a result. It is the duty of the psycho-anæsthetist to prevent 'psychic trauma' by suggestion by drugs and if necessary, by inhalation of nitrous oxide or cyclopropane. In America, earphones are often supplied to patients undergoing operations under local analgesia so that they can listen to recorded music.⁸ Their tastes are ascertained beforehand and selections from classical, semi classical and popular tunes^{*} can be supplied.⁹ Apart from its psychological aspects, pre anæsthetic fear has two physical consequences. Firstly, it increases the amount of adrenaline in the circulation and this is known to be a predisposing factor in the causation of ventricular fibrillation. Secondly the metabolic rate is raised which is the exact opposite to the ideal condition if a nitrous oxide-oxygen anæsthesia is to be superimposed.¹⁰

* Certain songs such as 'My Heart Stood Still' are not recommended

CHAPTER 20

PSYCHOLOGICAL ASPECTS OF ANÆSTHESIA AND ANALGESIA

Suggestion—Hypnosis—Personal Factor—Anæsthetic Outpatient Clinics—Anæsthetic Rooms—Recovery Rooms

THE psychological aspects of anæsthesia have not received much attention in the past but in the last few years have come greatly to the fore. The patient rightly regards a serious operation as one of the most important events in his life and not only appreciates real kindness and sympathy from his anæsthetist but is also in a state which readily responds to suggestion.

Suggestion

The late E. R. Wilson did a great deal to demonstrate the physiological basis of suggestion.¹ It has been shown² that there is localization in depth as well as on the surface of the cortical grey matter the three cell layers being (a) the supragranular or intelligence layer, constituting the conscious brain and governed by the laws of reason, argument, etc. (b) the granular or 'artistic' layer concerned with subconscious memory, music etc. (c) the infragranular or instinctive layer. The two lower layers together constitute the subconscious brain and obey the laws of reflex action which have been fully investigated by such workers as Sherrington and Pawlow. It follows then that if interference from the supragranular layer can be inhibited we are left with a subconscious brain which will blindly accept any suggestion made to it. The various means at our disposal for inhibiting the conscious brain are

- (i) Psychological methods e.g. by temporary distraction
- (ii) Certain drugs, e.g. barbiturates
- (iii) Sub-anæsthetic concentrations of narcotic gases

In recent years adequate sedation has been used almost universally before general anæsthesia for major surgery and thus the psychic factor has been largely eliminated (see Premedication). Even if preliminary narcotics are not used the anæsthetist can still render the induction of anæsthesia pleasant by suggestion and can even modify

The advantages of the method are the absence of toxic effects from drugs e.g. post anesthetic vomiting the retention of protective reflexes (e.g. coughing) and in obstetrics the fact that the foetus is unaffected.

The disadvantages are the uncertainty of results and the fact that only 20 per cent of patients can be placed in a sufficiently deep trance for major surgical operations to be carried out. Nevertheless, if in any given case there is a definite contra indication to general anaesthesia and if a local method is impossible it is worth having a trial run to ascertain if the patient is a good hypnotic subject.¹⁶

The methods of induction of the hypnotic state will depend largely on the practice of the hypnotist but the two most successful are (1) direct authoritative suggestion and (2) eye fixation combined with progressive suggestions of relaxation tiredness and sleep given in a monotonous and repetitive fashion. Any exotic techniques suggestive of charlatanry must be rigidly eschewed. The time of induction for the first session is usually about ten minutes and when the deepest possible trance has been attained, the patient is told that the whole operative field (with a considerable margin) is completely insensitive.



FIG 72 Incision in breast under hypnotic anaesthesia
(Mason *Anaesthesia*)

Hypnosis

Deep hypnotic suggestion can not only be used to modify general or local anaesthesia but can be used to produce analgesia itself. Hypnotism was practised by the Greeks in very early times and was re-introduced into civilization by Franz Anton Mesmer who renamed it mesmerism. About 120 years ago surgical operations were being performed in large numbers under hypnotic anaesthesia. For example, in 1819 Cloquet performed a radical mastectomy with dissection of the axillary glands. In 1843, John Elliotson published a booklet on numerous surgical procedures he had performed under hypnosis and in consequence was obliged to resign from the staff of University College Hospital. In 1847, Squire Ward amputated a leg and presented the case to the Royal Medical and Chirurgical Society. James Esdaile, working in Bengal, recorded no fewer than 300 major and 2000 minor operations on Indians. The term hypnosis is said to have been coined by John Braid of Manchester.¹⁰ When the anaesthetic properties of nitrous oxide, chloroform and ether were discovered they attracted the whole attention of the surgeons then practising and hypnosis lapsed.¹¹ This process was accelerated by the fact that many quacks exploited the financial aspects of hypnotism and highly reputable medical men suffered from being associated with it. During the second World War interest in hypnotic anaesthesia revived. For example, during the Japanese occupation of Singapore the stocks of anaesthetic drugs became extremely low and in the beginning of 1945 some success was achieved with hypnosis in minor surgery.¹² After the war considerable work was done on the subject and the technique can now be said to have attained complete respectability since the Psychological Medicine Group of the British Medical Association produced a favourable report on *The Medical Uses of Hypnotism*.¹³

The first matter to be considered before using hypnotic anaesthesia is the probability of the patient reacting favourably to it. It is now generally agreed that about 10 per cent of all patients are completely unaffected, that 35 per cent can be put into a light trance, 35 per cent into a medium trance and 20 per cent into a deep trance. The last is essential for major surgical procedures; a medium trance will suffice for minor operations while no analgesia is present in a light trance, although this state may be useful to allay anxiety before operation. The figures given above are almost unaffected by age, race and sex, although patients used to a life of discipline (e.g. service personnel, factory workers, nurses, etc.) respond more readily than those with analytical minds (e.g. scientists).

albuminuria and anemia. These should be referred to the appropriate departments for treatment before admission. Pre operative breathing exercises can be inaugurated if desirable and there is a good deal to be said for routine radiography of the chest. For example 1000 patients were radiographed at a clinic and 116 abnormalities were detected. Upon further investigation 29 operations were postponed.²¹ Dental sepsis can be diagnosed and treated before operation and a special diet prescribed for obesity, and in many other ways patients can be made fitter and less likely to have post operative complications. Beds can frequently be saved by not admitting patients who for the moment are unfit for operation.

Apart from physical examination, patients are encouraged to ask questions at these clinics and the whole pre operative technique is thoroughly explained to them. Their preferences and past experiences can be discussed with the anæsthetist and the most suitable methods of induction and maintenance of narcosis can be settled.²²

Anæsthetic Rooms

Reference has already been made to anæsthetic rooms. Although most hospitals are equipped with them, they usually fall far short of the ideal from the psychological point of view.

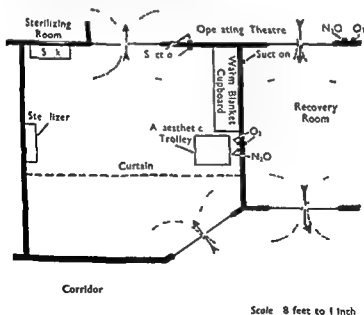


FIG 73 Plan of anæsthetic room for gynaecological theatre St Bartholomew's Hospital (Hewer *Brit med J*)

It is usually wise to mark out the skin area with a pencil so that the patient is left in no doubt as to its precise extent. At the termination of the required period the patient must be definitely awakened from his trance or unpleasant sequelæ may occur.

The Personal Factor

It is, perhaps, insufficiently appreciated how differently individuals react to identical stimuli, and, indeed the same individual varies from time to time. For example, such factors as a delicate upbringing, culture, education, artistic temperament, fatigue and cold will produce surprising variations in the response to suggestion and the amount of anæsthetics required.¹⁵

Dawkins has estimated that the average length of time necessary to produce anæsthesia with nitrous oxide in fair haired patients was fifty two seconds and in red haired ones sixty eight seconds.

Drug addiction affects the susceptibility of patients to anæsthetics. The difficulty in inducing anæsthesia in alcoholics is well known and this has been quantitatively estimated in mice.¹⁶ Addiction to morphine and cocaine however, *increases* the susceptibility of animals to ether anæsthesia.

It has been shown that while women are more likely to vomit after operation, men are much more prone to the severe forms of post operative complications.¹⁷

Special care should be taken with children as a badly given anæsthetic may affect their mental outlook for a very considerable time. Adequate premedication is most important while deception and trickery of any kind should be eschewed.¹⁸

The weather also has a marked influence. For example, the lower the relative humidity, the shorter is the induction time. Again, as the barometric pressure falls the induction time increases.¹⁹

The extreme variability of action of drugs such as the barbiturates on patients who appear very similar has already been noted. Considerations such as these show the futility of any fixed technique or dosage.

Anæsthetic Outpatient Clinics

In busy hospital practice the preliminary investigation of the patient from the anæsthetic aspect is often unsatisfactory and in Great Britain the anæsthetic outpatient clinic has been started by several hospitals with great success.²⁰ A general check up is carried out here including urine testing and hæmoglobin estimation. A certain number of patients will be found with glycosuria.

Induction of anæsthesia having been attained by an intravenous barbiturate, the curtain is then drawn back when the gas oxygen and other apparatus becomes immediately available



FIG 75 Mural painting anæsthetic room
(Hewer Nursing Mirror)

Many other considerations enter into the design of an ideal anæsthetic room apart from those features which affect the conscious patient. Space is important many existing rooms being far too small for comfort. It has been suggested that the minimum floor space for

The two essential features in order to secure a reasonable environment for the induction of anæsthesia are silence and an absence of alarming objects. Quietness is not always easy to attain but it may help considerably to eliminate talking when a conscious patient is present, and to make sure that no electric bells, buzzers, telephones or the clattering of instruments disturb the peace. The approach to visual sensations should be both negative and positive. As regards the former approach, all unpleasant sights should be prevented for example it should be impossible for a patient in the anæsthetic room to see into the operating theatre even if the connecting door

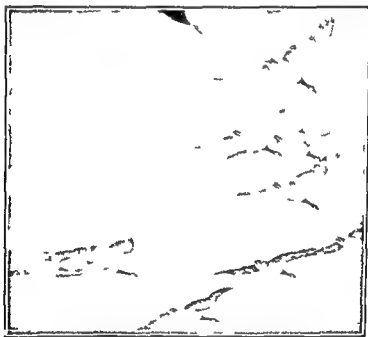


FIG 74 Photograph of ceiling of the anæsthetic room
(Hewer *Brit med J*)

■ open. Furthermore all anæsthetic apparatus and equipment should be kept out of the way until the patient is unconscious. One way of doing this is to divide the anæsthetic room into two compartments by means of a washable curtain running on a ceiling rod (Fig 73). The patient on his trolley is wheeled into the first compartment which contains nothing except ordinary domestic furniture.³ His attention can be directed to some pleasing object such as a painting.⁴ As he will be lying on his back pictures might be painted on the ceiling and the upper part of the opposite wall (Figs 74 and 75).

- 6 BUXTON D W *Brit J Anaesth* 7 69
- 7 PITKIN C P quoted by MAXON L II 1938 *Spinal Anaesthesia*
- 8 JARMAN R 1949 *Proc R Soc Med (An Sec)* Nov 5
- 9 *Lancet* (Annotation) 1940 June 24 1162
- 10 STEWART J D 1939 *Brit J Anaesth* Jan 41
- 11 HOLLANDER B 1932, *Proc R Soc Med (An Sec)* Feb 5
- 12 SAMPIMON R L and WOODRUFF M I 1946 *Med J Aust* March 23 393
- 13 Annotation 1955 *Brit med J* April 23 1019 and Suppl 190
- 14 MASON A A 1955 *Anaesthesia* July 295
- 15 STACEY J E 1935 *Brit med J* 820
- 16 ABREU B E and EMERSON G A 1939 *Anesth & Analges* Sept Oct 294
- 17 GORDII T 1949 *Proc R Soc Med (An Sec)* Dec 2
- 18 *Brit med J* (Edit) 1943 Dec 25 820
- 19 DAWKINS C J M 1938 *Brit med J* July 30 244
- 20 { LEE J A 1949 *Anaesthesia* Oct 169
- 21 { LODER R E and RICHARDSON H J 1954 *Lancet* June 5 1177
- 22 { LODER R E 1955 *Lancet* June 4 1150
- 22 HOWAT D D C and GREEN R A 1951 *Proc R Soc Med (An Sec)* May 4
- 23 OSTLERE G No 2 Vol 5 1950 *Anaesthesia* April 91
- 24 HEWER C L 1955 *Brit med J* July 23 257
- 25 WARD R 1949 *Design and Equipment of Hospitals* 213
- 26 MASON A A 1956 *Proc R Soc Med (An Sec)* Jan 6
- 27 JOLLY C and LEE, J ALFRED 1957 *Anaesthesia* Jan 149

an anæsthetic room serving one theatre should be 196 sq ft and that the corridor outside should be at least 7 ft wide so that trolleys can be turned easily into the door²⁵ Lighting is important and no bright lamp should be placed in the ceiling where it shines into the patients eyes An anglepoise or similar type of swinging bracket attached to a wall provides good lighting for barbiturate injections or for the setting up of intravenous infusions Ordinary electric bulbs are preferable to strip lighting as however they are coloured to simulate 'daylight,' in practice they distort colour values and render the detection of slight cyanosis difficult If an outside window exists a blind should be provided to facilitate difficult laryngoscopies and bronchoscopies The precautions against fires and explosions mentioned in Chapter 5 should be applied to anæsthetic rooms, e.g. the flooring should be of terrazzo or other material of suitable electrical resistance and all rubber components should be made of the anti static variety Nitrous oxide oxygen and suction are most conveniently supplied from wall plugs by pipe line the small cylinders on the anæsthetic trolley acting as an emergency reserve Full air conditioning is desirable so that the humidity and temperature can be controlled Until recently it has not been thought necessary in Great Britain to provide for temperatures lower than the outside air but the advent of induced hypothermia has now rendered essential the incorporation of a cooling unit in the air control system

Post-operative Observation Rooms

Although recovery rooms hardly come under the heading of psychological aspects their importance can be mentioned here They should be sited close to the theatres so that patients whose condition after operation is unsatisfactory can receive skilled and individual nursing attention under the immediate supervision of the surgeons and anæsthetists Facilities for suction oxygen administration bronchoscopy intravenous infusions etc should be provided When the patients are fit for transference to the ward the trolleys used should be of the tilting variety so that a suitable slope can be maintained if hypotension still exists²⁷

References

- 1 WILSON S R. 1927 *Proc R Soc Med* (An Sec) Feb 4
- 2 By CAMPBELL, BRODMANN BOLTON and others
- 3 HORNABROOK 1932, *General Practice* March 15 365
- 4 RAWLINGS N W *Brit J Anæsth* 7 127
- 5 CRAMPTON H P 1934 *Proc R Soc Med* (An Sec) 28 94

spite of these drawbacks, the system made progress and was adopted in some sections of the U S Army Medical Corps² More recently it has been used as the anaesthetic record system in the University of Wales³ It is said that the idea of punched cards occurred to Hollerith when he was travelling by train and watched a ticket collector punching the ticket margins in different positions according to the colour of the passengers' eyes and hair⁴ The object of this was to detect the transfer of tickets which was an illegal practice

In Great Britain a simpler type of combined chart and card index has been designed by Dr M D Nosworthy on the Copeland-Chatterton system⁵ and many teaching hospitals are now adopting it with a view to preventing the large mass of valuable data from



FIG 76 Sorting of marginal punched cards with long needle. The clipper for converting holes into slots is also shown (Maclean Smith *Lancet*)

CHAPTER 21

ANÆSTHETIC CHARTS AND RECORDS

IN previous chapters, there have been many references to blood pressure and pulse rate changes during anæsthesia and charts are usually kept during all prolonged operations. The form of these charts differs in various hospitals according to the type of case commonly encountered and they incorporate a varying amount of miscellaneous information relative to the patient, the operation and the anæsthetic. As a general rule this amounts to the sum total of anæsthetic records which are afterwards available for study.

Progress in anæsthesia is usually achieved either by the introduction of new agents or by improvements in technique. The difficulty is to determine whether a particular change in method is an advance or a retrogression. The only logical way to do so is to compare a large number of cases in which the change has been made with an equal number in which it has not, all other factors being the same. Under existing conditions this is impossible, and unless a new drug or method is quite obviously good or bad it takes a long time for anæsthetists generally to assess its value. Many instances could be cited of so-called advances which have gradually lapsed into oblivion in spite of enthusiastic write ups which were based more on wishful thinking than on hard facts. Commercial interests have been known to impart a more roseate hue to direct and indirect advertisements than has been subsequently found justifiable by the results obtained by the products concerned. It is true that individual anæsthetists have made careful records of cases done under new methods but a prolonged period must elapse before the number of administrations becomes really convincing while it is very difficult to compare the results with those of older techniques owing to differences in other factors.

The only way out of this *impasse* is for anæsthetists to keep *standardized* records which can readily be sorted out afterwards. The necessity for some such system was recognized in America some years ago and resulted in a modification of the Hollerith punch card system by the Committee of Records and Statistics of the American Association of Anæsthetists.¹ Unfortunately this necessitated a complicated and rather fragile card, the learning of an arbitrary code and an expensive mechanical sorting machine. In

spite of these drawbacks, the system made progress and was adopted in some sections of the U.S. Army Medical Corps.² More recently it has been used as the anaesthetic record system in the University of Wales.³ It is said that the idea of punched cards occurred to Hollerith when he was travelling by train and watched a ticket collector punching the ticket margins in different positions according to the colour of the passengers' eyes and hair.⁴ The object of this was to detect the transfer of tickets which was an illegal practice.

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FIG 76 Sorting of marginal punched cards with long needle. The clipper for converting holes into slots is also shown (Maclean Smith, *Lancet*).

TITRE										CHARTED PULSE & BLOOD PRESSURE										TOTAL PULSE & BLOOD PRESSURE										EFFECT OF PREPARATION										ANES. TECHNIQUES									
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<p>NOTES</p>										<p>CHARTED PULSE & BLOOD PRESSURE</p>										<p>TOTAL PULSE & BLOOD PRESSURE</p>										<p>EFFECT OF PREPARATION</p>										<p>ANES. TECHNIQUES</p>									
<p>POST-OPERATIVE EFFECTS</p>										<p>CHARTED PULSE & BLOOD PRESSURE</p>										<p>TOTAL PULSE & BLOOD PRESSURE</p>										<p>EFFECT OF PREPARATION</p>										<p>ANES. TECHNIQUES</p>									
<p>CHARTED PULSE & BLOOD PRESSURE</p>										<p>TOTAL PULSE & BLOOD PRESSURE</p>										<p>EFFECT OF PREPARATION</p>										<p>ANES. TECHNIQUES</p>																			
<p>CHARTED PULSE & BLOOD PRESSURE</p>										<p>TOTAL PULSE & BLOOD PRESSURE</p>										<p>EFFECT OF PREPARATION</p>										<p>ANES. TECHNIQUES</p>																			
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FIG 78 Back of Record Card showing blood pressure pulse rate charts etc (M D Nosworthy 3rd edition)

being wasted as has occurred in the past. A second and third edition of this card have now been issued with minor modifications.*

On referring to the illustrations, it will be seen that on one side of the card is a blood pressure and pulse chart specially adapted for operation records. The other side has a series of positive factors of all kinds each opposite a perforation near one edge of the card. At the time of operation the anæsthetist encircles the relevant positive factors, preferably in ink. This system eliminates all unnecessary writing. After operation the cards are completed and when the patient leaves hospital they are returned to the anæsthetic department where each is issued with a serial number which with the patient's name and operation performed, is entered in a card index. The holes opposite the encircled positive factors are then converted into V shaped slots with two cuts of a pair of scissors or with a special clipping device. Finally the corner of the card marked with the diagonal line is snipped off and all is ready for filing. It will be obvious by looking at the cut off corners that the cards are in proper order and not either upside down or back to front. Sorting of a pack is done with great ease by means of a knitting needle. For example suppose that it is desired to find the incidence of major pulmonary complications after partial gastrectomy performed under (1) cyclopropane and (2) spinal block over a year's period. The needle is first inserted into the upper laparotomy hole and raised. All cards now remaining in the pack will refer to patients who have undergone an upper laparotomy. These can then be subdivided into cyclopropane and spinal cases by needling the appropriate holes and finally each of the subdivisions is needed for major respiratory complications. The only hand sorting required is to separate partial gastrectomies from other upper laparotomies. It will readily be seen that the number of different investigations which can be carried out at any subsequent date is almost infinite.

Apart from facilitating research the writer has found the system of marginal punched cards to be of great value for teaching purposes. If an anæsthetic clerk has filled in a card correctly there is very little that he does not know about the administration of the anæsthetic and its immediate and remote effects upon the patient. The system also tends to focus attention upon the patient himself rather than upon anæsthetic apparatus and it is a certain prophylactic against the insidious disease of mind wandering. Subsequently it may be found that the card will furnish more general information to an investigator than the patient's bulky dossier of hospital notes.

The cards can be adapted for special purposes comparatively easily. For example, at the Brompton Chest Hospital they have been modified by pasting a printed slip of the commoner thoracic operations over the general list which is standard. Alternatively special cards embodying the same principle of sorting have been evolved for particular (e.g. thoracic) cases.⁷

In America the Nosworthy card has been enlarged and elaborated into the Chicago Anaesthesia Record which is also known as a 'key-sort Card'.⁸

In the opinion of the writers the universal adoption of some such system of anaesthetists would constitute one of the most important recent advances in the specialty, but it is a mistake to suppose that standardized records by different anaesthetists can be compared without reserve as it is impossible to eliminate the human factor completely.

The chief difficulty which has been encountered in practice has been the lack of clerical assistance. In busy hospitals, it may be physically impossible for the anaesthetists to find time to fill in the post-operative details of the records and to file and sort them. One solution to this problem is for the hospital statistical department to be responsible for the clerical and mathematical work involved, but it is essential that the co-operation of the anaesthetic department be obtained before any conclusions are drawn or grave errors can be perpetrated.

References

- 1 CHEVERS E M 1942 *Brit J Anaesth* 18 69
- 2 WANGEMAN C P 1941 *Anesthesiology* 2 179
- 3 MUSHIN W W 1953, *Proc R Soc Med (An Sec)* April 10
- 4 SMITH J M 1955 *Lancet* Oct 29 919
- 5 NOSWORTHY M D 1943 *Brit J Anaesth* 18 No 4
- 6 NOSWORTHY M D 1945 *Anesth & Analges* Nov-Dec 221
- 7 BALLANTINE R I W 1950 *Anaesthesia* Jan 44
- 8 CONROY W A *et al* 1948 *Anesthesiology* March 121

CHAPTER 22

OXYGEN THERAPY

Historical

PRIESTLY discovered oxygen in 1774 and soon afterwards suggested that it might be "peculiarly salutary to the lungs in certain morbid cases". Two years later John Hunter considered that inflation of the lungs with oxygen would be a valuable procedure in resuscitation. In 1780, Chaussier administered oxygen for asphyxia in the newborn infant and even devised a laryngeal tube for this purpose. The inhalation of oxygen for therapeutic purposes was popularized by Thomas Beddoes who in 1799 opened a Pneumatic Institute at Bristol with Sir Humphry Davy as director. The value of effective oxygen therapy was demonstrated by Haldane in the 1914-18 war and he developed it for the treatment of pulmonary oedema from gassing.¹

Physiological Principles

Man normally breathes air containing 20.96 per cent oxygen, and at sea level 95 per cent of his arterial haemoglobin exists as oxyhaemoglobin, whilst the dissolved oxygen in the plasma amounts to 0.24 per cent.

If a normal individual inhales pure oxygen, *all* the haemoglobin is present as oxyhaemoglobin and the plasma oxygen reaches 2.2 per cent. At rest the average adult requires about 300 ml of oxygen per minute but in strenuous exercise this can reach 3000 ml. At sea level this means that 90 litres of air must be passed through the lungs in each minute and in the same time the lungs must be perfused with 25 litres of blood or nearly 1 pint per second.⁴⁷

It has been shown by arterial puncture and blood analyses that in a great variety of pathological conditions the oxygen saturation of the blood is definitely diminished and that if this can be restored to normal, great improvement in the patient's condition results. This, then, is the object of oxygen therapy.

Toxic Effects

The next point to decide is whether the inhalation of high percentages of oxygen itself results in any harm. Paul Bert in 1878² and Lorrain Smith in 1899³ thought that the gas should not

be given in concentrations higher than 60 per cent. It was stated that rabbits subjected to nearly pure oxygen inhalation for some days eventually succumbed to pulmonary edema. This statement retarded the development of effective oxygen therapy, but it is now recognized that it is fallacious to suppose that a hypoxic patient will react in the same way as a normal rabbit.⁴ Furthermore it now seems doubtful whether the original experiments were conclusive. In order to elucidate this point, rabbits, guinea pigs and rats have been exposed to pure oxygen for sixteen hours per day. At the end of fifty consecutive days no ill-effects were observed. As regards the effect of the gas on healthy human beings, continuous inhalation of 100 per cent under normal pressure for twenty four hours (no mean feat!) sometimes produces temporary substernal distress but there is no significant change in pulse rate, blood pressure or blood counts.⁵ It can, therefore, be assumed that injury to the lungs from oxygen inhalation at atmospheric pressure is highly improbable.

At high pressures, however, the gas is not so innocuous. Paul Bert showed in 1878 that convulsions may occur when oxygen is breathed under high pressures. This subject is of importance to divers who can carry on much longer if oxygen is substituted for compressed air while a further advantage is that no bubbles of nitrogen rise to the surface to betray their presence to possible enemy observers. It has long been recognized in the Navy that it is dangerous for divers to breathe pure oxygen at a pressure of three atmospheres,⁶ although many healthy men can submit to this severe test for three hours without distressing symptoms. After this progressive contraction of the visual fields⁷ and the onset of convulsions may ensue. As a result of repeated exposures to the pure gas under high pressure some divers eventually develop an idiosyncrasy to it.⁸ The Admiralty Experimental Diving Unit carried out many experiments from 1942 to 1944 in preparation for attacks on enemy ships such as the *Tirpitz*. It was found that there was extreme variation in reactions and a much lower tolerance under water than in compressed air. For example convulsions have occurred at a depth of only 40 ft.⁹ Later work on animals showed that the onset of convulsions can be delayed by barbiturate narcosis and by chlorpromazine.¹⁰ While these observations do not assist divers they may be of great use in deep X ray therapy combined with high pressure oxygen (see later).

In certain morbid conditions the inhalation of oxygen at atmospheric pressure can give rise to toxic symptoms. During some phases of congestive heart failure due to chronic disease of the lungs

cor pulmonale, oxygen therapy can cause a sharp rise in cerebrospinal pressure occasionally with disastrous results. The reason for this is obscure.

The prolonged inhalation of high percentages of oxygen by premature infants is now thought to be unwise as it may tend to retrolental fibroplasia with ultimate blindness¹¹ (see Chapter 18).

Indications

The chief indication for the administration of oxygen is hypoxia due to any irremovable cause.

Hypoxia can be classified as follows¹²

(a) Obstructive. Mechanical respiratory obstruction may prevent an adequate amount of air from entering or leaving the lungs or may impede alveolar diffusion. This class will include such cases as obstructive goitre, glottic oedema and pneumonia.

(b) Non obstructive

(1) Increased metabolic rate as in acute thyrotoxicosis¹³

(2) Deficient circulation as in failing heart, shock etc (stagnant hypoxia)

(3) Deficient amount of oxygen in inspired air as at high altitudes. In aviation it has been found that added oxygen is beneficial even at such moderate heights as 10 000 ft¹⁴ unless a pressurized cabin is used (see later). The history of the conquest of Everest has emphasized the extreme importance of oxygen in high altitude mountaineering where the exercise involved adds the factor of increased oxygen consumption to a deficient intake.

(4) Decreased oxygen carrying capacity of the blood as in anæmia and in sulphonamide and carbon monoxide poisoning (anæmic hypoxia)

(5) Decreased capacity for using oxygen by the tissues as in extreme cachexia, cyanide poisoning and rheumatic myocarditis¹⁵ (histotoxic hypoxia).

Decreased respiratory movements from central and peripheral paralysis may cause hypoxia but here assisted or controlled respiration is indicated rather than an increase in the oxygen percentage of the inspired air.

One of the less pleasing amenities promised during the next war is the employment of nerve gases. They were discovered in 1935 by a German scientist, Schrader and although not actually used in the Second World War were found in the possession of the German

army during the Rhine crossing in 1944. For security reasons full details of these compounds cannot be given but it can be stated that at atmospheric pressure they are liquids with boiling points between 150°C and 250°C . The vapours are colourless, odourless and undetectable by natural means—a new instrument has, however, been developed which appears to be a reliable detector. Standard Service respirators give protection against these gases. These poisons are absorbed readily by the inhalation of vapour or by contact of the liquid with skin or mucosa. They are intensely toxic, and act by the inhibition of cholinesterase with consequent accumulation of acetylcholine. Acute poisoning leads to asphyxia with death from anoxia. The treatment is (1) large doses of atropine intramuscularly (e.g. gr 1/32) and (2) immediate and prolonged artificial respiration if the respiratory exchange is inefficient. The Ministry of Supply advocates a hand bellows for this purpose¹⁶ but intermittent oxygen inflation would be more efficient if the apparatus was available.

Oxygen therapy has also been used successfully in the treatment of such conditions as surgical emphysema, air embolism and acute distension of the stomach and small intestine¹⁷. The rationale of this procedure is as follows. Collections of gas trapped in the body consist chiefly of nitrogen. If the patient breathes pure oxygen for some time the partial pressure of nitrogen in the alveolar air will fall. The blood nitrogen concentration will also fall so that this gas will pass from any trapped collections to the blood and will be removed by the lungs.

Recently it has been shown that the sensitivity of certain tumour cells to irradiation is increased if the patient is breathing oxygen at high pressure¹⁸. The technique for this procedure is mentioned later.

Finally if foetal distress is diagnosed (such as a foetal heart rate of over 160 or below 100) the inhalation of high concentrations of oxygen by the mother nearly always causes temporary improvement in the condition of the foetus. Incidentally the very success of this treatment is a potential danger in that necessary action may be delayed⁵¹.

Sources of Oxygen Supply

Medical oxygen is usually supplied in cylinders at a pressure of about 2000 lb per sq in. Reduction of pressure may be affected in one or two stages. In hospital practice a battery of large cylinders is often located in a special cylinder room and the gas is piped to the wards or private rooms. In any such system it is essential that

the oxygen supply should be controllable by cut off valves situated in an accessible position outside the ward or room. The failure to observe this obvious precaution has resulted in many fatalities from fire.

Several modern hospitals in Great Britain now use liquid oxygen for their supply. The gas is liquefied at the factory and transferred to insulated globular tanks mounted on lorry chassis. These tanks are of about 14 000 cu ft capacity and have a blow off valve at the top so that as the temperature of the liquid (-183°C) gradually rises gaseous oxygen escapes to some extent. On arrival at the hospital the liquid oxygen is siphoned out into a large vaporizing tank which must be housed in a separate single storey building with direct access to a road. When the correct volume of liquid has been transferred the escape valve is closed and the pressure gradually rises the rate being controlled by immersion heaters in the water jacket of the vaporizer. The high pressure oxygen gas is then used to fill a battery of large cylinders which are permanently installed. The advantages of using liquid oxygen are the elimination of noise and the labour of off loading large cylinders from lorries and transporting them to the battery. The cost of the gas used is also somewhat reduced.

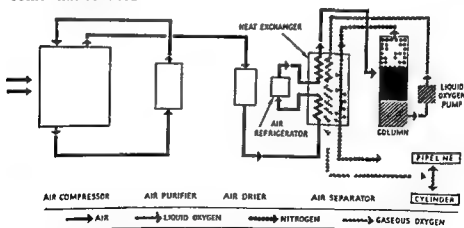


FIG 79 Diagram showing principle of oxygen generator suitable for hospital installation (The Butterley Co Ltd)

A third source of oxygen supply is the manufacture of the gas on the hospital premises. Oxygen generators abolish transport problems and can be divided into two main types. Large generators suitable for providing gas for medical wards and operating theatres require no chemical substances for their use and are merely dependent on

electrical power. The principle is a simple one as atmospheric air is liquefied by a powerful compressor and is then fractionally distilled. Nitrogen boils off first, the oxygen remaining as a liquid.

Small portable generators are extremely valuable in wartime and in places remote from cylinder filling plants. They have been used for some years on submarines and the principle of operation is the reaction of a mixture of chlorates with an iron catalyst in a closed chamber. In practice the chemical candles are ignited in the apparatus and the resulting oxygen is purified by passage through a solution of sodium hydroxide and hyposulphite. For safety reasons the generator should be placed outside the ward or operating theatre, the oxygen being led to the patient from a reduction valve.

Methods

Face-piece. If a patient has stopped breathing, his chest must be rhythmically inflated by oxygen under pressure. The emergency

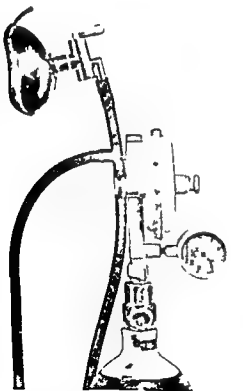


FIG 80 Oxygen Inflator (The Oxford) for patients who are not breathing

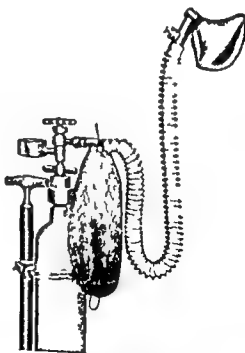


FIG 81 Oxygen inhalation attachment to cylinder (Magill's) for patients who are breathing but unconscious

oxygen valves on many types of anæsthetic apparatus are ideal for this purpose. Alternatively the special reducing valve shown can be attached to an oxygen cylinder. The screw at the side of the valve adjusts the pressure up to 45 mm Hg. The face piece is held firmly on the patient's face and the lever above it is turned to

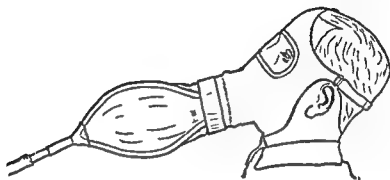


FIG 82 Emergency oxygen inverter made from Government respirator and football bladder (Marriott *Brit med J*)

ON. When the chest is expanded it is turned back and the elastic recoil of the thorax causes deflation.¹⁸ This sequence can be produced automatically by an apparatus such as the pneumatic balance resuscitator which in effect provides intermittent positive pressure.¹⁹

If the patient is unconscious but still breathing pure oxygen can be supplied from any gas oxygen apparatus or from a simple attachment to an oxygen cylinder as shown (Fig 81).



FIG 83 Nasal oxygen inverter (Christie *Lancet*)

In cases of emergency, an efficient oxygen inverter can be made from a civilian or civilian duty respirator and an Association football bladder size 4 or 5. A hole is cut in the latter opposite the inlet and is stretched so that the bladder overlaps the canister and is reinforced by a rubber band (Fig 82). The oxygen flow is adjusted so that the bladder is about half full at the end of inspiration.²⁰

The face piece method is obviously unsuitable for prolonged administration and is not well tolerated by a conscious patient.

Nasal Inhaler A nasal inhaler, such as is used for dental anaesthesia, can be employed with an intermittent flow apparatus. A mixture containing from 60 per cent to 100 per cent oxygen can be inhaled continuously provided that the patient keeps his mouth shut. The amount of rebreathing must be adjusted according to the per



FIG 84 B.L.B. oxygen inhaler
(Oxygen Therapy Equipment Ltd.)

centage of CO₂ desirable which is usually minimal. This type of inhaler can be worn night and day without much discomfort and even at high concentrations the oxygen consumption should not exceed 100 gallons per hour.

A specially modified nasal inhaler has been devised by R. Christie.

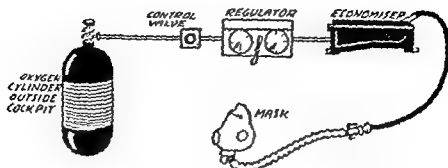
The extremely thin walled bag is made of gold beater's skin. Even if no special effort is made to breathe through the nose, good results are claimed at a flow of 3 to 4 litres per minute.¹

The B.L.B. inhaler is of similar type and was widely used during the late war. Either a nasal or oronasal mask can be used and a variable proportion of air is admitted by rotating a sleeve which uncovers one, two or three holes. Partial rebreathing takes place into a small rubber bag.



FIG 85 Polythene mask and latex bag in position (Kent Lancet)

An oxygen flow of 3 litres per minute with two air holes open

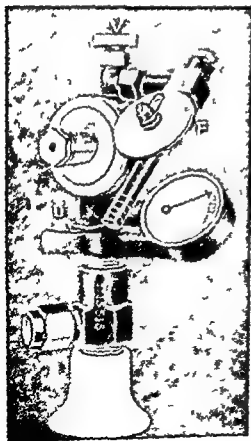


LAYOUT OF OXYGEN SYSTEM

FIG 86 Oxygen economizer
for aircraft pilots
(Whittingham Brit med J)

DIAGRAM OF
ECONOMISER →

→ FLOW FROM
REGULATOR



should give an alveolar concentration of about 40 per cent, while it should be possible to step up the oxygen percentage to 90 with a flow of 6 to 8 litres per minute and all holes shut.² Unfortunately this apparatus has several drawbacks. At low oxygen flows the resistance to breathing is considerable and the CO_2 concentration may exceed 2 per cent. Furthermore, the variation in oxygen concentration of the inspired air is as high as 18 per cent fluctuating with the depth of respiration.²² Various modifications of the B L B inhaler are in use²¹ such as the very light plastic mask developed for high altitude

FIG 87 Injector unit to supply adjustable mixtures of oxygen and air (M I E Co)

V Fine adjustment valve F Control disc D To humidifier (Cowan and Mitchell Brit med J)

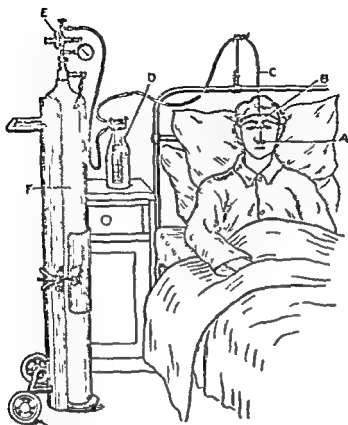


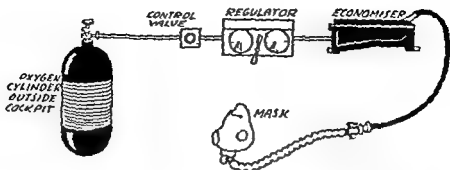
FIG 88 Apparatus for the administration of oxygen by nasal catheters (Marriott and Robson *Brit med J*)

flying and made of polythene (Alkathene ICI) (See Fig 85) The actual lay out of a suitable oxygen system for aircraft pilots is shown in Fig 86 This incorporates an economizer which shuts off the supply during expiration *

In order to obviate the disadvantages of flow meters an injector has been devised to fit on to the oxygen cylinder so that the desired mixture with air is obtained at the source of supply No flow meter is required and valves are provided so that no rebreathing occurs the bag simply acting as a reservoir ** It seems probable



FIG 89 Spectacle type nasal catheter carrier (Tudor Edwards)

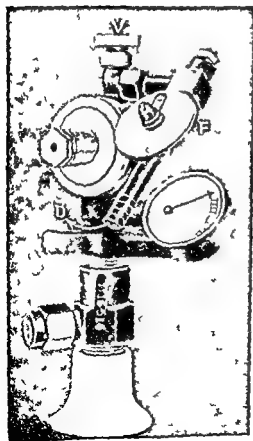


LAYOUT OF OXYGEN SYSTEM

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FIG 87 Injector unit to supply adjustable mixtures of oxygen and air (MIE Co)

V Fine adjustment valve F Control disc D To humidifier (Cowan and Mitchell *Brit med J*)

considerably reduced by combining a nasal catheter with a light aluminium box mask, as shown in Fig 91

A combined reducing and fine adjustment valve R leads the oxygen through a bobbin type flow meter F and humidifier W to the catheter which may be placed either 3 in inside the nose or outside it and directed against the middle of the top of the mask

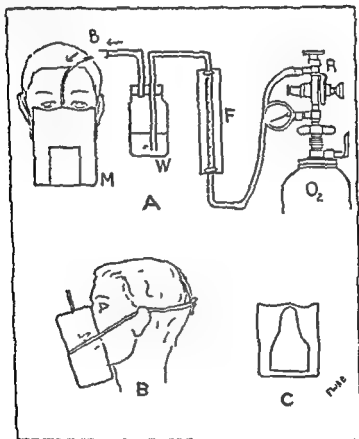


FIG 91 Nasal catheter combined with mask (Campbell *Brit med J*)

The opening for the nose and mouth is shown at C. The lower part of the mask is open. When the catheter is intranasal the alveolar oxygen at 4, 5, 6 and 7 litres per minute is given at 40 per cent, 50 per cent, 60 per cent and 70 per cent respectively. If the catheter is outside the nose the system is not quite so efficient.²¹ In an emergency a mask can be constructed quickly from light cardboard. The most recent type has the shoulders of the mask rounded off thus improving vision and making the apparatus more comfortable to wear.²

that some such arrangement will soon become standard practice

Nasal Catheter The *simplest* technique for effective oxygen therapy is undoubtedly by means of nasal catheters²⁸ Good results are obtained by using two small lubricated rubber catheters A (Fig 88) passed about 3 in inside the nose These are connected to the catheter carrier B fixed by a head band or a spectacle frame

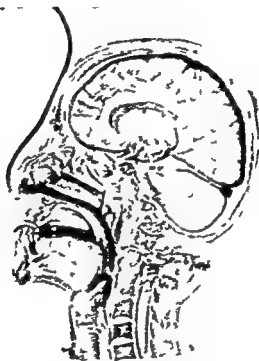


FIG 90 Sagittal section showing correct position for naso-pharyngeal catheter (after Waters)

(Fig 89) Thick walled pressure tubing C conveys the oxygen from a reducing valve E via a combined flow meter and humidifier D Gas flows of 4 to 16 litres per minute are stated to give oxygen percentages in the alveolar air of about 30 to 60 respectively and little difference is noted whether the mouth is open or shut²⁹

A more efficient technique is to use a single nasal catheter whose tip is in the oro pharynx. The appropriate length of insertion is the distance between the nostril and the tragus of the ear The catheter should be inserted while the oxygen is flowing to just beyond the estimated distance It is then withdrawn until swallowing no longer occurs³⁰ The rather large oxygen flow can be

considerably reduced by combining a nasal catheter with a light aluminium box mask, as shown in Fig 91

A combined reducing and fine adjustment valve R leads the oxygen through a bobbin type flow meter F and humidifier W to the catheter which may be placed either 3 in inside the nose or outside it and directed against the middle of the top of the mask

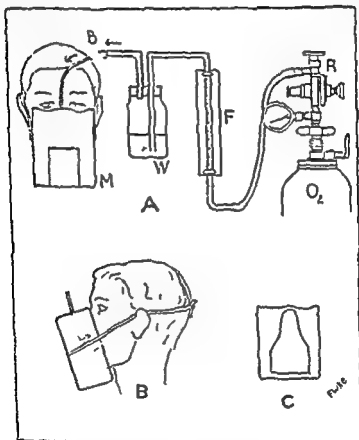


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It will have been noticed that most of the devices described require flow meters and reducing valves. In cases of emergency it is possible to treat two patients with only one flow meter and valve by using a special Y tube, the upper arms of which contain resistances consisting of pinhole openings in brass discs. The lower limb of the Y is connected by pressure tubing to the cylinder reducing valve while the two upper limbs supply the selected apparatus to the patient. A single flow meter is interposed in one circuit and each patient will receive practically the same volume of

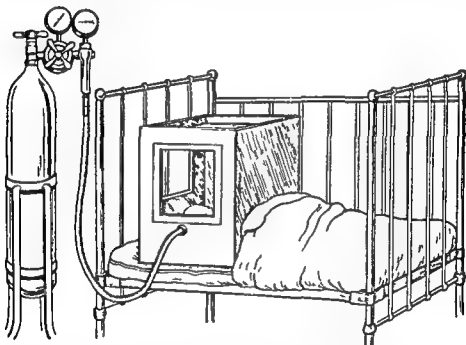


FIG 92 Oxygen box (after Burgess)

oxygen per minute. This idea might be of great use in Service or air raid practice, particularly if a large number of gassed patients had to be treated simultaneously with limited equipment.³³

Oxygen Box. An oxygen box is a simple and efficient device if no tent is obtainable. A box measuring about $28 \times 18 \times 18$ in. is used with either an open or closed top and with one side replaced by a curtain to fit around the patient's neck (Fig 92). An oxygen inlet is arranged near the bottom and a flow of 4.5 to 5 litres per minute gives a continuous concentration of 40 per cent to 60 per cent at the level of the patient's nose in spite of free upward diffusion with the top open. In the closed condition a flow of only 1 litre per

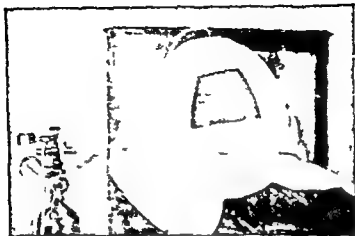


FIG 93 Typical motorless oxygen tent (Heidbrink)

minute will provide a concentration of 70 per cent oxygen after 3 hours. Adequate control of temperature can be obtained by the use of an ice container within the box.²⁴ The oxygen box is of great use for babies with asphyxia neonatorum and for premature infants²⁵ (see p 228).

Oxygen Tents. Oxygen tents are extremely useful for delirious patients and those who will not tolerate a nasal catheter or inhaler. Tents vary from simple mica hoods to those embodying extremely intricate mechanism. Fig 93 shows the simplest possible type in

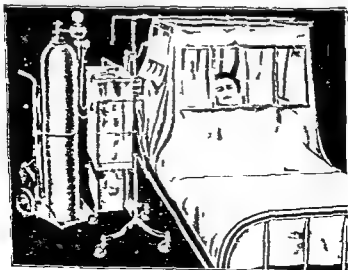


FIG 94 Simple type of oxygen tent for emergency use

which the gas enters the top of the hood by a jet which causes a pleasant breeze to play upon the patient's face increases the concentration near the nose, and helps to cool the internal atmosphere. If the flow meter is set at 5 litres per minute an oxygen concentration of 60 per cent to 70 per cent is obtained. An electric fan playing on the outside of the hood also assists in cooling. Carbon dioxide escapes through the rubberized fabric and should not exceed 1 per cent even after prolonged use. It is found, however, that with this simple type of tent moisture tends to accumulate and the patient after a time, feels confined and oppressed. Two minutes' relief after every half hour is generally given and the inhalation also has to be interrupted when medicine and food are administered.

For these reasons the more elaborate types of tent have been designed. Although of many different patterns these usually incorporate the following features:

(1) Positive ventilation is secured either by a pump driven by an electric motor or preferably by an injector on the oxygen feed²¹ or by the up and down movements of a small gasometer worked by an oxygen motor.

(2) Definite regulation of the temperature and humidity of the internal atmosphere is obtained by an ice chamber and thermostat. A hygrometer is usually incorporated and the relative humidity should normally be kept below 50 per cent.

(3) Regulation of the CO_2 percentage is secured by a soda lime chamber. If $\text{O}_2 + \text{CO}_2$ mixtures are desired this filter is cut out and CO_2 added from a separate cylinder.

(4) Provision is made for securing samples of the internal atmosphere for gas analysis. It is essential that the oxygen concentration be estimated from time to time as leakage is frequently a cause of ineffective therapy.²⁵

(5) The transparent windows must always be made of non-inflammable material such as cellulose acetate. Neglect of this precaution (e.g. the use of cellulose nitrate or celluloid) has led to disasters.

(6) The interior of the tent must be sterilizable.

The chief disadvantage of oxygen tents are the high cost of maintenance and the fact that some patients experience a type of claustrophobia when confined within them.²⁶ The cost of running tents can be considerably reduced by using commercial oxygen instead of the highly purified gas prepared for anaesthetic purposes. The only impurity in the former product is nitrogen and since an atmosphere containing less than 70 per cent oxygen

is usually employed, this is of no importance. The cost can also be cut down by having hospital wards piped for oxygen with a control valve near each bed as described earlier.

The remaining methods of giving oxygen inhalation are by means of Oxygen Chambers,³⁷ Oxygen Rooms, and Oxygen Wards.³⁸ These have never been popular in Great Britain but many hospitals in the United States and Canada were at one time equipped with them. They have now become obsolescent owing to the practical impossibility of eliminating the risk of fire. For example a slight fault in the electrical wiring which would merely blow a fuse in an ordinary room may give rise to a raging furnace in a few seconds if the atmospheric oxygen percentage is high and many patients have perished from this cause.

Technique of Oxygen Inhalation

The patient's condition should first be noted with special reference to cyanosis, air hunger, pulse and respiration rates, blood pressure and mental state. It must of course be realized that cyanosis is not necessarily a sign of anoxia. Pure oxygen should then be administered (in practice this will not exceed 98 per cent) until maximum improvement has taken place. If none occurs within eight hours the oxygen can be abandoned as useless. The oxygen percentage is then gradually reduced until the minimal amount is found which will maintain the patient's condition at the highest level. This concentration is continued until recovery from the disease permits a lower percentage to be employed. This process is continued until atmospheric air can be inhaled without deterioration in condition. This technique requires hourly charting of pulse, respiration and blood pressure for considerable periods but the dramatic results obtained when the treatment is scientifically carried out more than justify the extra trouble entailed.³⁹

It cannot be too strongly stressed that the patient must not have a cigarette lighter or matches in his possession and smoking by him or by visitors must be absolutely forbidden.

Technique of Oxygen Pressure Inhalation

It has recently been shown that the sensitivity of certain tumour cells to irradiation is increased if the patient is breathing oxygen at a high pressure.⁴⁰ A tentative technique has been worked out using a naval divers decompression chamber.⁴¹ The patient is anaesthetized with a method incorporating a barbiturate and chlorpromazine to

avoid convulsions (see earlier) Bilateral myringotomy is then performed to avoid tympanic rupture or hemorrhage into the middle ear After oral intubation, the patient is left to breathe spontaneously, his inspiration taking place direct from the chamber and his expira

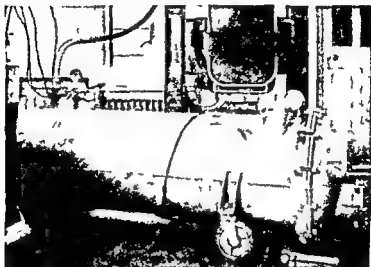


FIG 95 Oxygen pressure-chamber in position under X ray unit (Churchill Davidson *Lancet*)

tion passing through a carbon dioxide absorber The chamber is pressurized with oxygen to 3 atmospheres absolute (30 lb per sq in gauge pressure) the process taking about 10 minutes The necessary irradiation is then carried out and decompression effected in about 7 minutes Bends and caisson disease are uncommon with pure oxygen

Subcutaneous Administration of Oxygen

Oxygen has been administered subcutaneously for some years⁴¹ but recently doubts have been thrown upon its efficacy It has been shown that the efficiency of the lungs in saturating the blood with oxygen is from fifteen to twenty times greater than that of the whole subcutaneous region of the body At first sight therefore it seems problematical whether subcutaneous oxygen could have much value⁴ There is no doubt, however, that clinically considerable improvement in a patient's condition may ensue⁴² and in hemoptysis⁴³ and acute asthma⁴⁵ this method is said to yield good results Subcutaneous oxygen is also of definite value in acute inflammatory conditions where it is possible to inject gas into or over the affected part For example, many cases of intractable sciatica and other

forms of neuritis have been cured in this way⁴⁶ Oxygen has also been used successfully in injecting infected joints in order to promote synovial secretion and to prevent adhesions,⁴⁷ while infiltration for gas gangrene has caused spectacular improvement⁴⁸

Technique Some such apparatus as the oxygenateur of Dr Bayeux of Paris, is generally used, the average dose being 500 ml If the patient's circulation is poor it is advisable to distribute the gas by injecting 200 to 300 ml in different areas The dose should not be repeated until the gas has been absorbed, as shown by the absence of crepitations

Intravenous Administration of Oxygen

It is possible to inject intravenously about 10 ml of commercial medical oxygen per minute or 20 ml of the pure gas prepared by electrolysis of a 10 per cent solution of sodium hydroxide This is under 10 per cent of the basal requirement and the flow cannot be increased without risk of gas embolism Nevertheless improvement has been recorded in cases of pneumonia⁴⁹

Surface Application of Oxygen

The exposure of certain surface lesions such as chronic ulcers of the leg to an atmosphere of oxygen is said to be very beneficial⁵

References

- 1 HALDANE, J S 1917 *Brit med J* 1 181
- 2 BERT P 1878 *La Pression Barometrique* 764 Paris
- 3 SMITH L 1899 *J Physiol* 24 19
- 4 EVANS J H 1927 *Anesth & Analges* 6 57
- 5 COMRIE J H *et al* 1945 *J Amer med Ass* 128 710
- 6 DUDLEY S I 1935 *Proc R Soc Med (United Serv Sec)* April 8
- 7 BEHNKE A R *et al* 1936 *Amer J Physiol* Jan 1 436
- 8 BEHNKE A R 1941 *Anesthesiology* May 245
- 9 DONALD K W 1947 *Brit med J* May 17 and 24
- 10 CHURCHILL DAVIDSON I *et al* 1955 *Lancet* May 28 1091
- 11 { ASHTON N *et al* 1953 *Brit J Ophthal* 37 513
- 12 { PATZ A *et al* 1953 *Amer J Ophthal* 36 1511
- 13 Modified from *Index of Differential Diagnosis* by H French 1923 156
- 14 JOLL, C A 1935 *Brit med J* May 18 1050
- 15 BARACH A L 1937 *J Amer med Ass* May 1868
- 16 POULTON E P 1939 *Lancet* Aug 3 305
- 17 { Annotation 1952 *Brit med J* Aug 9 334
- 18 { Annotation 1952 *Lancet* Aug 9 286
- 19 FINE J *et al* 1935 *Amer J Digest Dis* Aug 361
- 20 MACINTOSH R R and PRATT C L C 1939 *Lancet* Jan 28 206
- 21 ADELMAN M H *et al* 1949 *Anesthesiology* Nov 673
- 22 MARRIOTT H L 1940 *Brit med J* Oct 19 519
- 23 CHRISTIE R 1938 *Lancet* Oct 15 880
- 24 BOOTHBY LOVEFACE and BULBULIAN 1938 *Staffs Mtgs Mayo Clinic* Oct 12

- 23 BARACH A L and ECKMAN M 1941 *Anesthesiology* 2 421
- 24 CARD W I 1944 *Lancet* Feb 5 177
- 25 KENT B S 1946 *Lancet* Sept 14 380
- 26 WHITTINGHAM H 1955 *Brit med J* Feb 5 305
- 27 COWAN S L and MITCHELL, J Y 1942 *Brit med J* Jan 24 118
- 28 BOURNE G 1922 *Lancet* 2, 23
- 29 MARRIOTT H L and ROBSON K 1926 *Brit med J* Jan 25 154
- 30 WATERS R M *et al* 1936 *Hospitals* March
- 31 CAMPBELL J A 1936 *Brit med J* June 20 1245
- 32 CAMPBELL, J A 1938 *Brit med J* June 11 1260
- 33 WRIGHT T M and CHRISTIE R V 1943 *Brit med J* March 11 287
- 34 BURGESS A 1933 *Anesth & Analges* Sept-Oct 220
- 35 { BARACH A L 1936 *J Amer med Ass* Feb 29 725
- JACKSON C R S 1951 *Brit med J* Nov 10 1129
- 36 JOLL, C A 1935 *Brit med J* May 18 1050
- 37 HILL, L 1933 *Lancet* Feb 18 384
- 38 DAVIDSON A E 1933 *The Modern Hospital* Feb
- 39 EVE F C 1939 *Brit med J* July 1 20
- 40 GRAY L H 1953 *Brit J Radiol* 26 609
- 41 BURKARD A F 1932 *Med World* March
- 42 SINGH I 1932 *Quart J exp Physiol* 22 193
- 43 SIMON O H 1934 *Anesth & Analges* Nov-Dec 233
- 44 LATVINE A 1934 *Brux Med* 23 219 (Dec)
- 45 EVANS J H and DURSHODWE C J 1937 *Anesth & Analges* July-Aug 211
- 46 BROWN H H 1938 *Brit med J* Dec 31 1390
- 47 HENSON E H 1936 *West Virginia med J* Feb 83
- 48 MACDONALD N M 1944 *Brit med J* April 1 470
- 49 SINGH I and SHAH M J 1940 *Lancet* May 18 922
- 50 *The Times* 1935 July 13 4
- 51 FITZGERALD T B and MCFARLANE C H 1955 *Brit med J* Aug 6 358

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- 23 BARACH A L and ECKMAN M 1941 *Anesthesiology* 2 421
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- 25 KENT B S 1946 *Lancet* Sept 14 380
- 26 WHITTINGHAM H 1955 *Brit med J* Feb 5 305
- 27 COWAN S L and MITCHELL J Y 1942 *Brit med J* Jan 24 118
- 28 BOURNE G 1922 *Lancet* 2, 23
- 29 MARRIOTT H L and ROBSON K 1926 *Brit med J* Jan 25 154
- 30 WATERS R M *et al* 1936 *Hospitals* March
- 31 CAMPBELL J A 1936 *Brit med J* June 20 1245
- 32 CAMPBELL J A 1938 *Brit med J* June 11 1260
- 33 WRIGHT T M and CHRISTIE R V 1943 *Brit med J* March 6 287
- 34 BURGESS A 1933 *Anesth & Analges* Sept-Oct 220
- 35 { BARACH A L 1936 *J Amer med Ass* Feb 29 725
- JACKSON C R S 1951 *Brit med J* Nov 10 1129
- 36 JOLL C A 1935 *Brit med J* May 18 1050
- 37 HILL L 1933 *Lancet* Feb 18 384
- 38 DAVIDSON A E 1933 *The Modern Hospital* Feb
- 39 EVE F C 1939 *Brit med J* July 1 20
- 40 GRAY L H 1953 *Brit J Radiol* 26 609
- 41 BURKARD A F 1932 *Med World* March
- 42 SINGH I 1932 *Quart J exp Physiol* 22 193
- 43 SIMON O B 1934 *Anesth & Analges* Nov-Dec 233
- 44 LATINNE A 1934 *Brux Med* 23 219 (Dec)
- 45 EVANS J H and DURSHORDWE C J 1937 *Anesth & Analges* July-Aug 211
- 46 BROWN H H 1938 *Brit med J* Dec 31 1390
- 47 HENSON E B 1936 *West Virginia med J* Feb 83
- 48 MACDONALD N M 1944 *Brit med J* April 1 470
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